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A PLEA FOR WIDER EMPLOYMENT OF THE ROENTGEN RAYS IN EXAMINATION OF THE LUNGS*

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Employment of the roentgen rays for examination of the lungs was one of their earliest applications to clinical medicine, and the progress of the method either was witnessed by, or is a familiar story to, most physicians. Today this test is demanded almost as a routine in hospitals and clinics everywhere, but in practice outside such institutions the method is used sparingly, far too sparingly in my opinion. It is true that in a majority of cases pulmonary affections can be identified clinically, and I have deep respect for the skill with which such diagnoses often are made. In too many instances, however, the symptoms, signs and ordinary laboratory tests are inadequate to make the differential diagnosis, to disclose complications, to determine the exact site and extent of the lesion, or even to establish or exclude the existence of disease. In such instances roentgenologic examination in competent hands, will seldom fail to give decisive information, and even when the clinical analysis is fairly complete and accurate the roentgenologic investigation usually will furnish valuable additional facts.

Of all the senses that of vision is most comprehensive and most dependable. Seeing is believing and the visual evidence supplied by the roentgenogram is far more convincing than inferences derived from the anamnesis and physical examination. In the normally transradiant lungs anatomic changes, however slight, are depicted in shadows that are almost as capable of diagnostic interpretation as a pathologic specimen. This does not imply that clinical examination should be superseded by the roentgenologic method, for symptoms and physical signs are primary indexes that can rarely be omitted from consideration, but they vary in diagnostic value so widely among diseases of the lungs, among different stages of disease and among individual patients that in most cases supplementary information is requisite for confident and complete diagnosis. Usually roentgenograms will furnish this information.

CLASSIFICATION OF PATIENTS

On the basis of clinical manifestations patients may be classified in four groups: (1) A group in which symptoms and signs are trivial, vague and inconclusive with regard to the existence of pulmonary disease. (2) A group in which the clinical manifestations are attributable to disease of organs other than the lungs and thus are often highly deceptive. (3) A group in which the clinical data are definitely indicative of a pulmonary affection but are indecisive as to its nature. (4) A group in which the anamnesis and physical signs point unerringly to a specific diagnosis, but often fail to disclose facts that are of practical significance. In all of these groups roentgenologic examination is either indispensable or desirable.

In the first group of patients, those who

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have few or no symptoms and none that are diagnostic, almost any of the multitude of pulmonary diseases may occasionally be represented, especially those in an early stage. Most conspicuous and common among them is early tuberculosis, for in a large proportion of cases the clinical examination is indecisive, either as to the existence of a lesion or as to its character. Here the roentgenologic examination offers extraordinarily valuable service.

Sampson and Brown[1] have said: "Today we have progressed to the stage where we call no man sound pulmonically until his stereoscopic roentgenograms have been found to be normal, and consider the roentgenologic study as much a part of the routine examination of the lungs as the stethoscopic, or, indeed, as the examination of the sputum." Again they have said: "The time is now upon us . . . . . when it will no longer do for us to wait for the development of clinical pulmonary tuberculosis before attempting to control it. We must discover which individuals have definite pulmonary lesions and endeavor so to direct their lives that the disease, undiscoverable by physical signs, to be revealed only by roentgenology, may make no further advances."

X-RAY FINDINGS

Roentgenologic manifestations of early tuberculosis in adults are of two principal varieties. The first and most common variety is a delicate conical or fan-shaped shadow resembling a caterpillar's web, in an upper lobe opposite the first or second interspace wih its apex toward the hilum and its base at the periphery of the lung. In the second variety the lesions have the same situation as in the first, but are irregular in shape and vary in diameter from 1 or 2 mm. to several centimeters. Their shadows may be discrete, or closely aggregated and are relatively faint, especially at their periphery. In both varieties the bronchovascular trunk leading to the affected region is accentuated.

Clinical recognition of hematogenous miliary tuberculosis of the lungs arising from a small focus in the lungs or elsewhere is scarcely possible. Roentgenologically the tubercles appear as myriad minute flecks scattered over both lungs and resembling the cut surface of a fine grained sponge. The picture is almost but not quite pathognomonic, for it must be distinguished from that of pneumoconiosis and of miliary metastasis.

Primary tuberculosis of children often gives rise to pronounced mediastinal or hilar lymphadenopathy as revealed by a widened median shadow or accentuation of the hilar shadow on the affected side. The Ghon focus in the parenchyma may be visible as a round or irregularly shaped shadow in any of the lobes, especially in their lower portion or in the hilar region. As a rule its appearance resembles that of coarsely ground glass, and this in association with the obvious lymphadenopathy serves to distinguish the lesion from bronchopneumonia or lobar pneumonia, although occasionally roentgenologic distinction is difficult.

During recent years the discovery and diagnosis of pneumoconiosis in its numerous varieties have become increasingly important. Not merely coal miners, quarrymen and cement workers, but all those engaged in dusty occupations, or who live in sandy regions in which dust storms occur are subject to this affection. Symptoms are absent or few in its early stages but the roentgenogram will reveal the deposits as a shower of discrete flecks in the lungs somewhat like those depicting miliary tubercles but slightly larger, denser, more discrete and less numerous in the apexes and bases. As the deposits increase and complications develop, the shadows become larger and more or less confluent, especially in the hilar region.

CLINICAL SIGNS

Occasionally the clinical manifestations are emphatic, but strongly indicative of disease elsewhere than in the lungs. Among such examples is spontaneous pneumothorax. The sudden, keen intrathoracic pain, the dyspnea and cyanosis might easily lead the clinician to suspect that their origin was in the heart rather than the lungs. But the roentgenologic signs are definite and diagnostic. The affected region, whether part

or all of a lobe or an entire lung, is brightly translucent and devoid of normal bronchovascular markings. In addition, if the pneumothorax is extensive, the heart and mediastinal structures are displaced toward the opposite side. Congenital cyst of the lung has a superficial resemblance to pneumothorax but distinction between the two usually can be made.

Not infrequently a small bronchiogenic carcinoma of the lung produces metastasis to the brain, and the symptoms are solely or predominantly cerebral.

In the third group, the patients whose clinical manifestations are definitely pulmonary but not specifically diagnostic, the rarer diseases are found. Included among such affections are congenital cyst, bronchiogenic carcinoma, Boeck's sarcoid, mycosis and many others.

CYSTS

Congenital cysts of the lung are indeed rare, for fewer than 200 cases have been reported, but doubtless many cases are overlooked and the affection requires more consideration than it has received. The cysts may be single or multiple, small or large, and situated in any part of either lung or in both lungs. They may contain air or fluid or both. Cysts filled with air only appear as transradiant areas in which no pulmonary markings can be seen, and the thin wall of the cyst often is depicted as a delicate linear shadow. In shape the cysts may be rounded or may conform in part to the boundary of a lobe, or, if multiple and closely packed, may be polyhedral. Cysts filled with fluid cast dense shadows that can hardly be distinguished from those of tumors. When the cysts contain both fluid and air, the fluid level surmounted by an air-filled dome is obvious, but distinction from other such cavities is necessary.

BOECK'S SARCOID

Boeck's sarcoid is an uncommon disease, the precise cause of which is not known. Pathologically it is characterized by the formation of nodules in the skin, thickening of the trabeculae together with areas of rarefaction in some bones, particularly the phalanges, and, in some cases, the development of fibrotic strands and nodules in the lungs. In typical cases roentgenograms of the lungs disclose dense bilateral linear shadows in the middle portion or lower half of the lungs, extending from the hilum well out toward the periphery. Interspersed among the streaky shadows are round dense shadows of miliary nodules, of various sizes. The largest is about 1 cm. in diameter.

CARCINOMA

Primary carcinoma of the lung now is considered invariably to be bronchiogenic in origin. Early carcinoma of a principal bronchus is characterized roentgenologically by a unilateral, hemispherical, triangular, or ill-defined shadow centered at the hilum. As a result of the usual obstruction the tributary parenchyma becomes atelectatic and correspondingly dense. Often atelectasis is the sole early manifestation, the growth being as yet too small to cast a detectable shadow. Carcinoma arising farther out in the bronchial tree begins as a single nodule, most often in an upper lobe. The growth is more or less spherical and its shadow is neither very dense nor sharply limited. As primary carcinoma in either situation progresses, complications occur, such as pleural effusion, metastasis and pyogenic infection, and the roentgenologic picture becomes more complex.

In the fourth, and probably largest, group of cases, those in which the clinical opinion is not open to question, roentgenologic investigation might seem to be ultrascientific and superfluous. But, however skillfully clinical examination may be carried out, it will seldom reveal the precise extent and site of pulmonary disease, or definitely establish or exclude the presence of all complicating factors. Practically all typical cases of common diseases, such as bronchiectasis, tuberculosis, pneumonia, and so forth, fall in this group.

On roentgenologic examination in patients with bronchiectasis the cylindrical, saccular or spherical dilatations of the bronchi are usually evident. When the sacs are multiple, grouped and filled with fluid, their dense shadows often resemble those of a bunch of grapes. When the sacs are empty,

the rarefied shadows depicting them are equally characteristic. If peribronchiectatic inflammation exists, the shadow of the inflammatory zone will be apparent. Sometimes, also, the roentgenogram discloses a foreign body, the presence of which was not suspected.

When it is definitely known that a patient has tuberculosis, the activity of his lesion is an important element in the case. Clinical indexes as to this factor, especially when the lesion is minimal, are often indefinite and require roentgenologic aid. The shadow of an active focus is hazy and ill-defined; that of a healed lesion is dense and sharply margined.

In moderately advanced cases of tuberculosis the prognosis depends largely on the degree of reactive fibrosis, and this can be determined with the roentgen rays.

Thus far only chronic diseases have been considered. In most cases of acute disease encountered at the office or in homes roentgenologic investigation seems to be unnecessary or impracticable, for the diagnosis is likely to be obvious or the patient is too ill to be sent out for the examination. But even in such cases, when the patient has pneumonia, localized pneumonitis, atelectasis or infarct, roentgenologic examination usually is well worth while.

Lobar pneumonia is depicted typically at the very beginning as an enlargement of the hilar shadow together with accentuation of the bronchovascular markings on that side. Progressively the shadow extends further out into the lung, usually into an upper or middle lobe, becomes dense and more or less homogeneous, and at least part of its margin coincides with the border of the lobe. When resolution of the lesion is beginning, its shadow becomes mottled with clear areas and its periphery recedes. Clearing continues until all shadows disappear.

Bronchopneumonia at its onset is characterized by enlargement of the hilar shadow like that of early lobar pneumonia. Later multiple discrete or partly fused shadows, variable in size, shape and density, and clustered about a bronchus appear further out in the parenchyma. Bronchopneumonia most often attacks lower lobes, does not respect lobar borders in its invasion and is often bilateral; all of these features are marks of distinction from the lobar variety.

When the history indicates that a foreign body has been aspirated, roentgenologic examination is not likely to be omitted, but sometimes, as in children, such aspiration is not known to have occurred. Metallic and other dense bodies can readily be discerned with the roentgen rays, but non-opaque bodies are not directly demonstrable. However, both varieties are likely to cause some degree of bronchial obstruction. If inspiration only is obstructed, the tributary segment of lung becomes atelectatic and dense. If the foreign body acts as a valve and permits inspiration but not expiration, the segment becomes emphysematous and brightly transradiant.

SUMMARY

Illustrations of pulmonary diseases and their roentgenologic characteristics could be multiplied indefinitely, for there are few patients in whom the method fails to be of service. But there is no need to lengthen a story that is already familiar. In the interest both of clinician and patient I have hoped merely to make those engaged in general practice more keenly conscious of the roentgen rays and their practical value. It seems to me that we may look forward confidently to a day not far off, when an efficient roentgenologic examination of the lungs will be applied as a routine in the diagnostic study of virtually all patients.

REFERENCE

1. Sampson, H. L., and Brown, Lawrason: Correlation of clinical and roentgenological observations in pulmonary tuberculosis, Radiology, 22:1, 1935.

DISCUSSION

Dr. I. L. Robbins (New Orleans): I would like to ask that you tell us briefly of the several occasions you remarked about when the x-ray man was able to diagnose the conditions of activity in the lungs. Tell us what are the finger prints of activity as far as the x-ray man is concerned in pulmonary disease. Also tell us if the x-ray man arrived at the diagnosis of pulmonary lesions by shadows.

Do you claim that there is anything distinctly different or unique as far as shadows of tuberculosis are concerned, pulmonary, as contrasted to other pulmonic lesions or influenced by the site of

lesions and various other factors in consideration, especially the history of the case and etiologic findings?

Dr. P. R. Gilmer (Shreveport): Dr. Kirklin has brought us a resumé of the conditions and findings which he has encountered, which were presented beautifully and all of us, I am sure, have gotten a lot out of his paper.

There is one case he showed which I do not pretend to take issue with as to diagnosis, but will ask him to elaborate on that case. It was the third case, where a solitary large shadow was seen in the lung, with complete absence of all clinical and physical signs. I would like to ask whether a stereoscopic examination was made, and whether or not that might not have been fibrous plaque left from previous pleurisy with fluid formation.

Dr. Kirklin (In conclusion): There is no doubt in my mind that in a high percentage of cases the competent roentgenologist can determine whether or not there is activity in the tuberculous lesion of the lungs. Clinical data are sometimes helpful in interpreting x-ray shadows in the lungs but they do not change the character of these shadows or the type of pathology that produces typical shadows. If the roentgenologist is familiar with these various types of pathology he should have no difficulty in making the correct interpretation. It is our practice to make stereoscopic films of the chest of practically every patient who comes for routine general examination.

WARREN STONE*

JOSEPH V. SCHLOSSER, B. S.†

Warren Stone was born at St. Albans, Vermont, February 3, 1808, the youngest of three children. His father, Peter Stone, died before he was born, and his mother, Jerusha Snow, from whom he inherited self-reliance and a remarkable physique, was forced to supplement the deficiencies of the scanty schooling of the day. His education conducted in part by tutors, and in part by his mother was entirely inadequate, which served to hamper him in later life. He left his rural home to become a private pupil of the eminent physician and surgeon of Keene, New Hampshire, Dr. Amos Twitchell, under whom he received the greater part of his extensive knowledge. He then attended the Medical School of Pittsfield, Massachusetts, graduating with the degree, Doctor of Medicine, in 1831. He began his practice in Troy, New York, and while there diagnosed the first case of cholera to enter the U. S. among immigrants fleeing to Troy from Quebec and Montreal. It was this experience that served him in good stead in later epidemics. Opportunities for practice being few in Troy, and looking to the South for his future home, he embarked for New Orleans on the brig "Amelia", in October of the same year. During the voyage a severe storm arose, necessitating the hatches being fastened down for several days, crowding 108 passengers in the stifling space between decks. On the third day there was a cry that a man was dead and Stone investigated and found 25 of the passengers in the second stage of cholera. The presence of disease and the poor condition of the ship following the storm necessitated its beaching on Folly Island off the coast of Charleston, where a rigid quarantine was set up. Physicians and supplies were sent to the island from the city of Charleston, but the physicians soon were broken by their exertions and requested relief from duty. Dr. Thomas Hunt was then appointed to take sole charge of the work. Dr. Stone remained on the island administering to the sick. There he and Hunt formed a friendship that was one of the most fortunate incidents of Stone's life, and one that was to lead to a momentous undertaking. While still on the island he became ill of cholera and was attended by Dr. Hunt. Following his illness, another vessel was chartered, the ill-fated "Amelia" having been burned, and he landed in New Orleans in December, 1833, at a time when concomitant epidemics of yellow fever and cholera were raging. He was sick and had insufficient clothing to protect him from the cold, and had but a single picayune in his pocket, which he later humorously said he preserved for the sake of a nucleus of his fortunes.

Because of the epidemic which carried off 6,000 people in a population of 50,000 and the great need for medical help, his proffered services were accepted by Dr. Cenas, who secured for him an appoint-

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†From the History of Medicine Society Tulane University of Louisiana, School of Medicine.

ment in a very subordinate position in the new Charity Hospital which had just been built. A few months later his good friend Hunt moved to New Orleans, and in August, 1833 became house surgeon at Charity Hospital. Hunt, in a letter to the Board of Administrators, warmly commended a promotion for Stone. A short time following the recommendation Hunt resigned to follow out his plans of founding a medical school, and Dr. John H. Harrison was elected in his place as house surgeon with Warren Stone as assistant house surgeon. Stone served in this capacity from 1833 to 1835, when upon the retirement of Dr. Harrison he became house surgeon, serving in this capacity from 1835 to 1839. At the expiration of his term he served as visiting surgeon until his retirement in 1872, in all serving the hospital 39 years.

Medical education was at a very low ebb at this time. Colleges were a product of private enterprise, organized as stock companies and composed of medical graduates depending solely upon the number of medical students and the amount of their fees. The Medical College of Louisiana, the foundation of the present Tulane University was just such a stock company, the product of the imagination, enthusiasm, and energy of Dr. Hunt. Dr. Stone was intimately associated with the college from its inception in 1834, serving the first few years as demonstrator of anatomy, lecturer in anatomy, and finally was elected to the professorship in 1837, on the petition of the admiring classes. He also served in the position of lecturer in surgery and in 1839 was appointed Professor of Surgery upon the resignation of Dr. Luzenberg, which post he held until his resignation in 1872.

In 1839 at the expiration of his term as house surgeon at Charity Hospital, Dr. Stone in connection with Dr. William E. Kennedy founded the Maison de Santé, one of the earliest private hospitals in America. This later formed the nucleus for the organization of Hotel Dieu. It was here that he lost his eye as a result of an inflammation incurred while treating a patient. It was a source of disfigurement and discomfort to him the rest of his life. Upon the founding of the Hotel Dieu, the Sisters of Charity went to that institution and the Maison de Santé became Stone's Infirmary. It was never a financial success.

Dr. Stone was a man of large physique, his huge frame being surmounted by a massive head and a face of imposing features with thick, bushy eyebrows, a firm mouth, and prominent jaw. Genial and frank though not hilarious, benevolent and charitable to a fault, he never turned a patient from his infirmary for inability to pay. He abhorred pretension of every kind, and was oblivious of fashionable convention of dress. Stone was a remarkably close and accurate observer and possessed a superior knowledge of anatomy, but was not didactic and never hesitated to admit possible error. His memory was unsurpassed and though he read little, he had a wide clinical experience and never forgot what be observed. He used no notes or memoranda, and it is said he could recall patients, even the most transient, describe their symptoms, and even remember the medication they received.

As a surgeon he was conservative, methodical, and industrious, a good anatomist, a keen diagnostician, and prompt though not graceful in execution of surgery when necessary. His resources were unbounded. He never seemed more cool or self-possessed than when suddenly confronted by some surgical emergency. He enjoyed the distinction of introducing the use of surgical anesthesia in New Orleans despite the antagonistic feelings of the profession, both locally and nationally. He conclusively demonstrated its worth by amputating a leg under ether anesthesia. He was the first to cure an aneurysm by compressing the artery, and successfully treated a patient with aneurysm of the vertebral artery by ligation and excision of the sac. He was the first to utilize rib resection to secure drainage in abscess of the liver and empyema. At the outbreak of the Civil War he was appointed Confederate Surgeon-General of the State of Louisiana, and served at the battles of Bull Run and

Shiloh. It was here his conservatism as a surgeon was manifest, saving many a limb that would have been unnecessarily amputated. He was a close friend of Jefferson Davis and his strong backing of the Confederacy caused his imprisonment at Fort Jackson for some weeks. He treated the Federals with the same regard as he had shown his own soldiers and because of his prominence was soon released. Stone acted as attending physician to Gen. Sherman, and when Gen. Grant was thrown from his horse at New Orleans he was called in to attend him.

As a lecturer he was discursive rather than systematic, and he was very apt to be led off the beaten paths by an interesting case. He wrote but little, being deterred by his inadequate early education. Some of his better known contributions are: Treatment of Wounded Arteries; Ligation of Common Iliac Artery; Inflammation; Pulmonary Tuberculosis; Amputation of the Mammae; Ligature of the Femoral Artery; Hernia and Obstruction of the Bowels.

Dr. Stone died at Baton Rouge, December 6, 1872, as the result of Bright's disease and diabetic gangrene, following a long period of illness. As a mark of respect the District Courts were adjourned and the flags of shipping stood at half mast for the funeral.

In the words of Judge Alexander Walker "A great man he truly was—great mentally, morally, and physically."

THE EFFECT OF HIGH TEMPERATURE ON BODY FUNCTIONS*

A SYMPOSIUM

THE REGULATION OF BODY TEMPERATURE

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Mammals and birds, so-called warm-blooded animals, possess efficient mechanisms for maintaining a constant body temperature against extreme changes in environmental temperature.[1] The regulation of body temperature is one of the best examples of homeostasis. The powers of adjustment to high and low external temperatures are greatly restricted as one grows older, and the homeostatic mechanisms are less and less able to adapt the organism to temperature variations in the external environment as age progresses in the later decades.[2] The body temperature represents the balance struck between the heat produced by the tissues and that lost to the environment. The heat produced in the body is a by-product of metabolic processes and the amount is far in excess of the usual requirement for keeping the body warm. Nature has provided means in the skin and lungs for the removal of the excess heat and for the maintenance of an optimal internal temperature.[3]

A fundamental requirement is the ability to adapt constantly to the ever changing physical environment, which tends to upset the temperature equilibrium of the body. Under the usual conditions of life these adjustments are so easily accomplished that a normal person is unconscious of the complex physiologic processes at work. Discomfort arises when thermal equilibrium is impossible or when abnormal reactions are involved in the transitional period of adaptation. The ability to adapt varies widely; metabolism, peripheral circulation, and physical and subjective symptoms show great variability in different persons under identical environmental conditions. Sensations of heat or cold are not due to change in internal body temperature but largely to changes in the temperature of the skin.

PHYSIOLOGY

In the brain-stem there are centers that coordinate the multitude of functions involved in heat regulation. When the cen-

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ters are aroused efferent neurons transmit activating impulses from the central nervous system to the various organs of the body controlling heat production and heat loss for the restoration of normal body temperature. When the external temperature rises, the blood vessels of the skin dilate by reflex vasomotor action. More blood flows through the skin, thus raising its temperature and therefore the heat loss. The rise is least in the skin of the forehead and trunk and greatest in the extremities, particularly the feet.[4, 5, 6] If this method of cooling is not sufficient to prevent a rise of body temperature, the sweat glands are thrown into action. In this way thermal equilibrium may be re-established if the external temperature is not too high.

On exposure to cold, a reverse action takes place. The skin vessels constrict and the blood supply is curtailed in order to conserve body heat. If this response fails to prevent the body temperature from falling, there is an increase in metabolism, effected partly by voluntary contraction and partly by stimulation of the adrenals and thyroid.

A rise in temperature causes an increase in circulating blood volume; the blood is diluted by fluid drawn into the circulation from the tissues, chiefly the skin, muscles and liver. Blood is expelled from the spleen. At low temperatures the blood volume is reduced, the blood becoming more concentrated as shown by an increase in the percentage of blood solids. These changes in blood volume are of paramount importance in the regulation of body temperature. The minute volume output of the heart is also increased when the temperature rises, and diminished when it falls.

Heat loss from the human body takes place by radiation to surrounding walls and objects, by convection to surrounding air, and by evaporation of moisture from the lungs and skin. Under comfortable conditions radiation usually accounts for between 46 and 60 per cent of the total heat loss, convection for from 15 to 30 per cent, and evaporation for from 20 to 30 per cent. The variation in loss through these different channels depends largely on physical and physiologic factors, the former including the temperature of the air and walls and the body surface exposed. The physiologic factors include the state of health, metabolic rate and vasomotor control. At usual indoor temperatures the average normal radiation loss is approximately 50 per cent, convection loss is 25 per cent and evaporation loss 25 per cent. About 9 per cent of the total heat loss takes place by evaporation from the lungs and upper respiratory passages, and 16 per cent by evaporation from the skin. Insensible perspiration is largely diffusion of water through the capillaries and is independent of sweat gland activity. Sweating is an emergency process that comes into play in warm environments or during muscular work. Insensible perspiration increases gradually with the environmental temperature up to a certain critical temperature between 86 and 90° F. at which point visible sweating breaks out.

There are important sex differences in temperature regulation, the skin temperature of the female being higher in warm atmosphere and lower in cold than the male. Women do not begin to sweat until the environment reaches a temperature two degrees above the threshold for sweating in men and the amount of sweat is less.[7]

The temperature and character of the objects towards which the body radiates are just as important as the temperature and radiating properties of the skin. This is one of the problems of modern air conditioning in which great strides have been made along purely engineering lines but in which knowledge of human requirements is incomplete and controversial. The nature of the basic physical and physiologic principles have been stated and are being studied by several investigators.[3, 6, 7, 8, 9, 10, 11]

Within a range of temperatures frequently referred to as the "comfort zone" (73° to 86° F.) the regulation of the dissipation of heat is accomplished and controlled chiefly by the sympathetic nervous system through the production of vasoconstriction or vasodilatation of the peripheral vascular systems of the upper and lower extremities.[5] The activity of the sympathetic sys-

tem is in a state of continual fluctuation of tone. The fingers are richly provided with arterial-venous anastomoses the function of which is to effect large changes in the amount and distribution of the blood flow. In some 500 determinations of the flow in the fingers Burton[4] found a range of from 1 c. c./min./100 c. c. of tissue up to a maximum of 90 c. c./min./100 c. c. of tissue. Average values over a five minute period lie between 15 and 40. In a person living for several days in a hot and humid room the maximum flow increased during five days of acclimatization from 96 to 122 c. c./min./100 c. c. In a cold room the minimum flow decreased in three days from 16 to 1.8 c. c./min./100 c. c. This tremendous range of flow represents not a metabolic need of the tissues but the mechanism for temperature regulation by elimination of body heat. Physical regulation of body temperature is accomplished, not by the setting of the peripheral blood flow to an appropriate steady level, but by the adjustment of the average of a flow which is fluctuating rhythmically between high and low values.

In the "zone of comfort", or zone of vasomotor control, there is, in addition to the loss of heat by radiation and convection, the loss of evaporation (insensible loss) which remains practically constant and accounts for about 25 per cent of the total loss of heat from the body. Above about 86° F. there is maintained maximal dilation of the peripheral blood vessels and the internal temperature of the body is kept constant by the increased secretion of sweat which occurs above this temperature. Considerable evaporation of the increased production of sweat will occur at temperatures above 88° F. if the humidity is low. Fairly high relative humidities may cause small increments in the skin temperatures of various regions of the body, particularly of the fingers and toes, because the loss of heat by evaporation of water from the skin and lungs is much reduced under relatively high environmental temperatures and humidities.

It is obvious that the nearer the temperature of the environment comes to that of the blood the smaller is the amount of heat which can be lost by radiation and convection. At an air temperature of about 98.6° F. heat loss by these means must cease. At higher air temperatures than this, the body, were there no other means of cooling available, would actually gain heat. Through the secretion and evaporation of sweat and the exhalation of water vapor large quantities of heat are lost to the body and its temperature can, for this reason, be maintained constant when the atmosphere (dry) has a temperature far above that of the blood.

It is to be emphasized that evaporation from the body surface, as from the surface of any material composed largely of water, occurs quite independently of sweat secretion. It has been shown for human subjects in whom sweat glands were congenitally absent, that some 16 grams of water per square meter of body surface may be lost hourly in this way. This represents a total daily heat loss of about 400 calories for a man of average size (surface area 1.8 square meters).

The rate of evaporation of water is influenced inversely by the degree to which the atmosphere is already saturated with moisture, that is, by its relative humidity. Sweat which is not evaporated but simply drips from the skin, of course, does not increase heat loss. For this reason the sweating mechanism for the elimination of heat is badly crippled when the relative humidity is high.

Humidity of the air has practically no effect on loss of heat by convection and, until quite high, does not appreciably affect loss of heat by evaporation.[5] Even a large rise in the percentage humidity of a room at 68° F. makes very little difference in vaporization, which at this temperature accounts for only 25 per cent of the total heat loss. The dissipation of heat from the body is dependent chiefly on environmental temperature and is little influenced by relative humidity when the person under test is in the basal metabolic state, lightly clothed and at rest in still air.

Sweat is a weak solution of sodium chlo-

ride (0.2-0.5 per cent) in water with traces of other salts and urea. According to Du Bois,[9] inhabitants of northern climates possess two or three million sweat glands, those who dwell in the tropics about a million more. They are man's safety valves in hot weather and there is no one human attribute of more importance than the ability to sweat skillfully.

The sweat glands are under the control of the sympathetic nervous system but are cholinergic, not adrenergic. The usual stimulus to sweat secretion is a rise in blood temperature which exerts its effect in two ways, firstly and more importantly, upon the nervous centers, and secondly, reflexly by stimulation of heat receptors in the skin. The sweat response to a rise in temperature is abolished by sectioning the nerves to a part and is not dependent upon the circulation. In the initial stages of muscular exercise sweating is due apparently to the discharge of impulses from the motor cortex. It occurs before there is any change in rectal temperature. Later on, the effect of a rise in body temperature comes into play. In a man (indoor clothing) at rest, visible sweating usually commences at an air temperature between 86° and 90° F. Sweating may be induced by the experimental stimulation of the hypothalamus and there are spinal centers also.

ENVIRONMENTAL TEMPERATURE

One of the most potent influences in increasing heat production is a low environmental temperature. In man (naked), the metabolic rate commences to rise when the air temperature falls to around 68° F. The external temperature at which the physical mechanisms are inadequate to maintain a constant body temperature, and must be aided by increased heat production, is called the critical temperature; it varies with the amount and nature of the clothing. The muscular tissues and the liver, wherein numerous chemical reactions are carried out, are the main sources of the body's heat. The rise in metabolism which results from a fall in atmospheric temperature is effected through an increase in tone of the skeletal muscles and in some instances by fine involuntary contractions, for example, shivering and chattering of the teeth. Food, especially protein, through its specific dynamic action, is an important factor in the chemical regulation of body temperature.

The heat regulating centers in the hypothalamus are influenced in two ways—reflexly from the skin and by the temperature of the blood flowing through them. The maintenance of a constant body temperature is due to the delicate balancing of activity of two quite distinct physiologic mechanisms. One, commonly spoken of as the mechanism of "heat loss" or "heat dissipation" prevents undue rise of body temperature by facilitating the dissipation of heat and possibly also, by reducing metabolic heat. The other mechanism commonly spoken of as that of "heat production", prevents undue fall of body temperature by increasing the heat produced, as well as by restricting the dissipation of bodily heat. It has been assumed that the various peripheral structures concerned in each reaction are controlled by the same central mechanism. During the past several years evidence has accumulated which indicates a certain structural and functional independence of these two mechanisms of heat loss and heat production and they have been separated by appropriate operation and stimulation. In the intact body these two mechanisms control each other antagonistically and thus balance each other.[12]

HYPOTHALAMIC LESIONS

The farther back hypothalamic lesions are placed the greater is the impairment of the ability to keep the body temperature up to the normal level; the posterior (sympathetic) portion particularly prevents chilling, or particularly activates heat production. Contrariwise, the anterior hypothalamus (parasympathetic) is not essential for adequate regulation against chilling, but damage to it causes serious impairment in the ability to prevent overheating; the anterior portion particularly prevents overheating, or particularly activates heat loss.[12, 13] Removal of the sympathetic renders an animal unduly sensitive to cold.

It is also possible that the thyroid and

adrenals are stimulated, and that the calorigenic effects of their secretions play a part in the thermoregulatory responses. Exposure to cold causes an increase in the rate of the denervated heart. It has also been reported that the serum taken from an animal exposed to cold raises the metabolism of a second animal into which it is injected. If the first animal has been thyroidectomized the effect upon the metabolism of the second animal is not observed. It may be mentioned in this connection that the temperature tends to be subnormal in suprarenalectomized or thyroidectomized animals, in Addison's disease, and in cretinism.

INDIVIDUAL ACCLIMATIZATION

In addition to the ability to adapt constantly to the changing environment is the ability to become adapted or acclimated to extremes of hot or cold weather. It is common experience that the initial discomfort becomes less after the first few days of exposure, and the "comfort zone" of temperature is between different limits according to the acclimatization of the individual to winter or to summer temperatures. There must exist slow physiologic adaptations, involving circulating blood volume, partition of heat loss, minute volume output of the heart, velocity of blood flow, which are superimposed upon the rapid mechanisms of adjustment to temperature, such that the acute reactions to temperature are slowly modified in their range and effectiveness.[11]

Adolph, Dill, Hall and Edwards[14] studied acclimatization to desert conditions at Boulder City, Nevada, and compared the results with those obtained in Boston. The conditions of climate were such as to allow maximal rates of evaporation without sweat appearing upon the skin, mean shade temperature of 95° F., maximum of 110° F. The mean humidity was 12 per cent and varied between 6 and 30. Several of the adjustments which occurred as the subjects became accustomed to life in the hot, dry climate, appeared to take place immediately, such as, increased evaporation, diminished urine output and increased intake of water. Among the slower adjustments were marked increases in rates of sweating, increased dilution of sweat, extending over a period between one and 10 days in different subjects, and an increase in the rate of ingestion of water. During residence in the dry desert, output by evaporation may increase eight-fold or more while the concentration of sweat diminishes. Sweat tends to become more concentrated as it becomes more profuse. In other words the more active the sweat glands, the less effectively is the chloride ion held back. But after several days of exposure to hot and dry conditions the concentration of sweat, at the same rate of sweating, for the same amount of exercise or work is only one half that at the beginning. Thus, one of the adaptive processes by which man becomes inured to high temperatures consists, in part, of the diminishing salt content of sweat. After acclimatization man will lose less salt and the satisfaction of his thirst will keep the volume and concentration of his body fluids more nearly optimal. Possibly heat exhaustion and certainly heat cramps are, in consequence, less apt to occur. Some subjects, however, remain susceptible to heat cramps because their sweat glands do not hold back salt effectively. Even after several weeks at Boulder City one of the subjects put out the equivalent of his daily salt intake in three hours of strenuous exercise. The study of "heat exchanges", that is the rate at which heat is gained and lost, is important because it is a plausible assumption that human endurance in hot climates is possibly limited by the rates of heat dissipation, great accumulations of heat in the body being thus prevented. At rest under the desert conditions, radiation may add heat to the body faster than heat is produced chemically. It was observed that in exercise, along with high rates of heat production, radiation and convection added heat, so that the loss by evaporation alone exceeded heat production. Recovery from exercise required one to one and a half hours for the dissipation of the heat that had accumulated. Evaporation rates were not limited by the evaporating power of the air in any of the conditions studied, since the skin stayed dry; the air was capable of

taking up moisture faster than the moisture was presented to it. One may think of the sweat glands and their control as limiting the amount of moisture that is presented for evaporation. The ability of diverse individuals to withstand desert conditions appeared to vary largely with the rates of sweat production, for with increased sweat production there was less heat accumulated in the body. Subjects who did not accumulate much heat were more comfortable, felt more fit, and were able to continue exercise for longer periods. Even they, however, were limited in the rates at which they could do work for any considerable time.

One limitation to work may be the rate of sweating. The changes with acclimatization, measured in rates of evaporation and in rate of heat accumulation in the body are significant factors in enduring a given hot atmosphere.

SUMMARY

Dill, in his book on Life, Heat and Altitude[15] points out that successful adaptation to high temperatures consists in the following: (1) An ability of the sweat glands to "learn" to secrete enough water to cool the body; (2) an ability to secrete this water with minimal amount of chloride, so as to conserve extracellular water and prevent depletion of sodium chloride; (3) an ability of the circulation to supply enough blood to the periphery to be cooled; and (4) the successful integration of these abilities to permit maximum loss of heat with minimum rise in body temperature.

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EFFECTS OF HIGH ENVIRONMENTAL TEMPERATURE ON THE HUMAN BODY

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A consideration of the effects of heat can be conveniently divided into the immediate effects that may be observed after a short exposure to high environmental temperature and those developing after prolonged exposure to such temperature, in other words, hot climate. Inquiring into both aspects of this problem, it is seen that little exact investigation has been done, and on many aspects there is lack of understanding. Accordingly, it will be necessary to leave many problems unsettled at points where information is much needed by the physician.

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EFFECTS OF LONG CONTINUED EXPOSURE TO HIGH ENVIRONMENTAL TEMPERATURE AND ACCLIMATIZATION

The ease or difficulty of body heat loss has been considered by Mills[1] as the significant factor in the effect of climate on man. This factor dominates the levels of internal combustion activity. Voit,[2] Rubner,[3] and Sundstroem[4] also found a relation between external temperatures and internal combustion rate in man and animals. Oxygen consumption increases as surrounding temperatures fall. The reverse, that is, decline in oxygen consumption with rising seasonal mean temperature levels, has been reported by various observers. Gessler,[5] Kunde,[6] Nakagawa,[7] Griffith,[8] and Martin,[9] all found a lower oxygen consumption in the summer months as compared to the winter months. Many of these observations were made under carefully controlled basal conditions. The decline in metabolic rate varied from 6 to 15 per cent. Seasonal fluctuations, according to Mills,[10] are probably widest in the middle and lower temperate regions where there is considerable depressive heat, but also, where the cold is not severe. Since environmental temperature influences the resting metabolism of man, it is argued that it has a more important effect upon the working metabolism or activity combustion of the individual. It is calculated that about 80 per cent of the combustion energy liberated through muscular activity must be dissipated from the body as waste heat, as our bodies are only about 20 per cent efficient as energy machines. Still further, it has been shown by McClintock and Paisley[11] that even this efficiency becomes less as the working metabolic rate rises. This produces the paradox that in what he does, the more active northerner is less efficient, so to speak, per stroke than the more sluggish southerner. In other words, even though the output of work per unit of time of the southerner is low, he is the more efficient from a machine point of view than the energetic northerner. Lee[12] finds that in addition to the excretory activity of the thyroid being reduced in conditions of heat, there is also a suppression of the activity of the suprarenal medulla, and he considers that this is one of the chief factors of acclimatization. Another effect is decreased appetite, which is a frequent accompaniment of exposure to heat. It is suggested that this is related to the decline in metabolic activity brought on by the heat.

Mills[10] found that in the 1934 heat wave there was a marked fall in blood pressure level and oxygen consumption even in those people showing no symptoms of heat stroke or prostration. Such a change happens in a minor degree in people going from temperate regions to the tropics. Animal experiments with rabbits were conducted by Mills with three groups of animals, which were in a cold room, a hot room, and were changed from one to the other, respectively. At the end of this time, they were tested for their ability to endure heat. The animals from the hot room withstood the heat the longest time, those from the cold room next, and those that had been changed daily from hot to cold were the least resistant. These experiments were interpreted as adaptation to constantly high temperatures rendered animals more resistant to excessive heat than is seen in adaptation to changeable environment. Logically then, the differences in susceptibility to excessive heat shown by individuals from varying locations are dependent upon those differences in metabolic activity and heat adaptation that have been set up in their own particular communities. Therefore, "the character of the climate determines in a large measure the general heat sensitivity of the population." It is further seen that the population as a whole is more affected by severe heat in the beginning than it is by similar heat at the end of summer. Such a change takes place in some degree during an individual heat wave, as it is seen that heat stroke tends to come at the end of such a wave.

The results of these metabolic effects on disease states must be stated with caution. Bean and Mills[13] find that coronary occlusion occurs more frequently in winter than in summer in the north temperate region studied. They find the same in regard to

heart failure in arteriosclerotic and rheumatic patients. Syphilitic heart failure fails to show any such relationship. It is presumed, however, that these observations contrast severe cold with summer warmth, rather than with tropical heat. Kligler[14] considers that tropical heat enhances the susceptibility to intestinal infection by retarded gastrointestinal secretion, by anhydremia and reduced gastrointestinal motor activity, and by a state of avitaminosis—particularly B, which follows the depression of the appetite.

THE IMMEDIATE EFFECTS OF HEAT

Prickly heat: In regard to frequency of the immediate effects of heat, the well-known condition of prickly heat, or lichen tropicus, or heat rash, is, as you know, the most common. Its clinical features are so familiar to this group as not to require description here. Explanation of the etiology and pathology, however, is not nearly so satisfactory. The cause of the condition is variously ascribed to exposure to excessive moist heat. Infancy, obesity, debility, overclothing, and a tendency to hyperhidrosis are regarded as predisposing. Immoderate use of alcohol renders one especially susceptible. Histologic study by Robinson[15] indicated that the papules were due to an inflammatory exudation around the orifices of the sweat ducts, but not of the sweat glands. Pollitzer[16] described vesicles occuring in the rete mucosum. The inflammatory reaction occludes the sweat duct. It is not clear in any investigation of this subject what the origin of the exudating process really is.

Heat urticaria: Less common, but of importance to many patients, is the urticaria produced by heat. This may be local or general. It is observed that many people have hives after a very hot bath or unusual exercise. This is the generalized type, and here the work of Grant, Pearson and Comeau[17] showed that the lesion was produced by the release of acetylcholine at the termination of the cutaneous nerves. In addition to the generalized type, there is a localized form of urticaria in which the wheals result from the direct action of the heat on the skin.[18]

Prophylaxis and therapy of skin effects of heat: The prevention of lichen tropicus and heat urticaria is obvious in type, and that is avoidance of exposure to such environment. When this is not possible, effort should be made so that exposure will be progressively increased and not be suddenly excessive at one time. The treatment of the lesions, once they have become established, is palliative and should consist of soothing lotions followed by the application of a bland powder, such as corn starch.

THE IMMEDIATE SYSTEMIC EFFECTS OF HIGH ENVIRONMENTAL TEMPERATURE

In the effort to maintain uniform body temperature in the presence of a high environmental temperature, the body is called upon for functional adaptations. Consequent upon these efforts, three conditions are known to develop among certain individuals exposed to this environment. These are: Heat cramps, heat exhaustion, and heat stroke.

Heat cramps: The condition now recognized as heat cramps was described in 1878, pertaining to conditions in the gold mines of Virginia City, Nevada. Attention to this condition for its clinical importance began about twenty years later, and the term is defined (Talbott)[19] as referring to painful spasms of the voluntary musculature following muscular activity in a high environmental temperature.

The incidence approximates that of heat stroke. The attacks are relatively common in the summer months in industrial centers in association with boilers and similar surroundings. Commonly, it is noticed in the steel industry and in the casting industry.

Although temperatures are high throughout the year, there is a distinct seasonal summer incidence. The humidity is of significance, but except in so far as it has some bearing on the effective temperature, it is secondary to the degree of heat itself. Predisposing causes are the use of alcohol, associated with insufficient food, mineral salts, water, and vitamins. Another predisposing factor is a recent attack of heat cramps not adequately treated. Other fac-

tors are those that have a definite but imponderable effect on general resistance, such as sleep, rest, irregular habits, and similar hygienic influences.

Symptoms: While at work on a hot day, or possibly hours later, the patient is suddenly seized with a violent pain associated with spasmodic contraction of various groups of muscles. Such spasms may occur in the muscles of the abdomen, arms, and back. The pain is severe. The patient may writhe and twist, and between spasms may toss about restlessly. In the space of five or ten minutes, the cramps recur and continue to the point where the patient may become exhausted.

Examination of the patient shows fibrillary twitchings of the muscles, violent contraction following manipulation of the muscles. The temperature is rarely above 100°, and the highest recorded is 104° F. The cardiovascular system is apparently not affected nor is the respiratory. It is the general impression that when sweating returns the cramps disappear.

Chemistry and morphology of the blood: Talbott[19] found the protein of the serum increased. The highest concentration observed by him was 10.6 grams per cent. He regards this as due to dehydration and probably not related to the pathogenesis of the disease. Concentration of the hemoglobin was correspondingly increased. He found that there was a loss of water from the red cells themselves of from 1 to 5 per cent.

Changes in the concentration of chloride and sodium were constantly present, and this has been found by other authors. Investigations by various authorities have shown reduction in chlorides varying from 2 up to 17 per cent. There is no significant change in the blood sugar. Non-protein nitrogen is elevated according to various observers. The amount of elevation is usually about 10 to 25 mg., but may be three times that. The red blood count is increased in proportion to the hemoglobin. The white blood cell count is increased, the usual counts being about 10,000 to 15,000.

Urine: The urine shows a diminution in the amount of chlorides during the attack. Chloride reappears in the urine one to three days after recovery.

Diagnosis: The diagnosis of heat cramps has to be made after considering other possibilities, such as those of an acute abdomen, strychnine poisoning, tetany, hysteria, and nocturnal cramps. It is not usually difficult when a correct history is obtained and the patient is seen experiencing the widespread muscular contractions of such a severe character.

Pathogenesis: Consideration of the data of various investigators indicates that heat cramps result from a diminished amount of sodium chloride in the blood, and probably, in the tissue spaces, and that when the critical level of sodium chloride is reached in the working subject muscle cramps appear. Consideration of other factors, such as disturbances of carbohydrate metabolism and acid base equilibrium, and kidney insufficiency, reveals that changes in these factors are insufficient to produce the effects mentioned.

Treatment: The patient should be put at rest. Sodium chloride and fluids should be supplied at once. The environment should be made comfortable. If facilities are available intravenous injections of normal physiologic saline should be given immediately. If able to take fluids and salt by mouth, one gram of sodium chloride should be given every hour until 5 grams have been ingested. It is common to observe that cramps subside as quickly as the effects of salt have been obtained. Should circumstances require it, morphine, ¼ gr. by needle may be used, but this is slower in giving relief than salt. The patient should be continued at rest for several days, and possibly, for a week.

Preventive treatment: The environment of the workers should be made as comforable as possible. Water should be supplied freely to those workers who of necessity must labor in high temperatures. Salt should be supplied in one of several ways: by using 1/10 of 1 per cent salt in the drinking water, or by the use of tablets

taken with the water or at meals. Attempts should be made to supply 5 to 10 grams of salt per day, depending on the duration of the heat exposure.

HEAT EXHAUSTION

In this condition, the patient who has been exposed to an excessively high environmental temperature becomes weak, complains of vertigo and dimness of vision. Excessive sweating is usually present. Examination reveals a state of shock, a weak, rapid, thready pulse, low blood pressure, normal or subnormal temperature, uneven respiration. The skin is clammy. The sphincters may be relaxed. The temperature may be subnormal, normal, or slightly above normal, if taken by rectum.

Examinations of the blood in this type of heat disease, by Heilman and Montgomery,[20] showed an average blood sugar of 64 mg. per 100 c. c., and a blood specific gravity of 1.062. This specific gravity figure was the same as that of controls. Plasma chloride concentrations showed changes, but not in proportion to the depletion of the blood sugar. Observations by McLain and Montgomery[21] showed a tendency to blood concentration, decrease in blood sugar, and lowering of alkali reserve.

Treatment: The treatment is essentially that of shock, with the use of 5 per cent glucose infusions. If the temperature is subnormal, external heat should be applied, but in this situation, the patient should be watched to prevent sudden hyperpyrexia. Coramine and adrenalin may be useful, as in other forms of circulatory collapse.

HEAT STROKE

Heat stroke tends to come in the summer months. It is found towards the end of a heat wave. It may develop in persons exposed to high external heat in- or out-of-doors. There seems to be a predilection for it to appear in infancy, or beyond the age of seventy. Individuals in the period of middle life are more resistant than those at extremes. Alcoholism is regarded as being contributory. Direct exposure to the rays of the sun does not appear to be an important factor. In a study made by Ferris, Blankenhorn, Robinson and Cullen,[22] 44 patients suffering from heat stroke were studied in the Cincinnati General Hospital. Only nine were doing work which required moderate physical effort. Thirteen of the patients were employed in working but not exposed to an environment warmer than that of the average person. Seven patients were less than 50 years old; the majority were 60 to 70 years old.

Prodromal symptoms: Certain patients complain of weakness, dizziness, nausea, and occasional attacks of fainting, two or three days before their collapse. Headache is common. There is a feeling of oppression by the heat.

Clinical manifestations: The patient may collapse suddenly and may or may not go into unconsciousness. During the onset, there may be a state of great nervous excitement simulating an acute mania, with laughing, crying, talking incoherently, and aimless and uncontrolled struggling with those about them. The skin is hot and dry. It is commonly reported that the patient's collapse follows a period in which he perspired normally, then suddenly ceased to perspire. There is a maculo-papular rash over the body which is frequently purpuric. Respiration is deep and stertorous. The face is flushed. The muscles are flaccid. There may or may not be incontinence. The acutely ill patients are unconscious or stuporous. Those with temperature of 106° or less are usually conscious. The blood pressure at the onset is elevated and declines as the clinical condition gets worse. With the increased blood pressure, there is a high pulse pressure. The pulse is rapid and may become irregular.

Chemistry of the blood: Examination of the blood in this condition by various investigators has revealed an increase in the serum chloride; and by others, no significant changes from the normal. In other respects, the state of the blood has not been found significantly different from that of controls.

Pathogenesis: The pathogenesis of this condition is not understood. It has been variously suggested that it is an exhaustion of the adaptation mechanism or an exhaus-

tion of the sweat producing system, a form of cardiac failure, a form of infection, and a disturbance of the heat control center. Noteworthy findings at autopsy, according to McKenzie and LeCount,[23] are edema and congestion of the brain, edema of the lungs, cloudy swelling of the liver, kidneys, and myocardium, and petechial hemorrhage of various mucous membranes. These findings are in agreement with the report by Wilson,[24] who found extensive hemorrhage under the endocardium of the left ventricle and septum in one patient dying of heat stroke and in three patients dying after fever therapy.

Treatment: The patient should be removed to a cool place with free circulation of air, all clothing removed, and covered with a wet sheet upon which there is a free flow of water and a steady stream of air from an electric fan or from manual efforts. While this is in process, fluids should be supplied by infusion or by a duodenal tube. The temperature should be reduced as rapidly as possible to about 102°; stimulants and other measures, such as recommended in heat exhaustion, may then become needed.

Prognosis: In 44 patients reported by Ferris, Blankenhorn, Robinson, and Cullen,[22] 17 or 39 per cent died. Of the 39 patients who lived long enough to obtain some form of treatment, 12 or 30 per cent died. None with a temperature below 107° died.

THE EFFECTS OF AIR CONDITIONING

Inquiry into the effects of air conditioning on the human organism is just beginning. The change from hot air into cold air in summer is more of a tax on bodily adaptation than in winter, because in winter the excursion into cold air is protected in advance by additional clothing. The rate of change rather than the degree of change appears to be the important thing. Mills[25] considers that the difference between outside hot and inside cool temperature in summer will have to be determined by observations for each locality. The further one progresses into tropical heat, the smaller should be the difference. The differences should be less in the latter part of the summer than in the early part of the summer. He suggests that in the Gulf area not more than 8° F. difference should be allowed. Where heat periods are brief and cool weather is common, a 20° difference might be tolerated, that in most of the northern United States a 12° difference is suggested.

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FULMINATING MENINGOCOCCAL SEPTICEMIA WITH BILATERAL SUPRARENAL HEMORRHAGE

REPORT OF TWO CASES*

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REVIEW OF LITERATURE

The first proved case of fulminating meningococcal septicemia (the so-called Waterhouse-Friderichsen syndrome) was reported by Andrews[1] in 1906 and occurred, curiously, in a 53 year old male; the disease is most frequent in very young children. The patient, who had previously been perfectly well, died 12 hours after the onset of symptoms, and a pure culture of meningococcus was isolated from the blood and from the spinal fluid, though no microscopic evidence of meningeal inflammation was found. Intracellular diplococci were also observed in the granulocytes of the antemortem blood smear.

The first presumptive case of this disease had been reported in 1894 by Voelcker,[2] who made a questionable diagnosis of an unusual type of smallpox. A previously healthy two year old child had suddenly become ill, with high fever and a purpuric rash, and had died within 24 hours. The only significant postmortem finding was bilateral suprarenal hemorrhage. No bacteriologic procedures were carried out. In 1901 Little,[3] on the basis of four personal cases and a study of eight other cases reported in the literature, stated that "the symptoms in all of these cases . . . are so similar and so constant as to form quite a recognizable clinical group." He was unable to demonstrate an infectious agent in any of his personal cases.

Waterhouse,[4] in 1911, reported a rapidly fatal case of suprarenal hemorrhage accompanied by skin purpura, and reviewed the 15 cases of this syndrome previously reported in the literature. He was unable to isolate any organism from the spinal fluid, spleen or suprarenal glands. Friderichsen,[5] in 1918, reported two proved cases of the disease and reviewed the 28 previously reported in the literature. Like Waterhouse, he pointed out the constancy of the clinical picture in this rapidly fatal form of meningococcal septicemia with suprarenal hemorrhage and an apparent absence of meningeal inflammation.

Glanzmann,[6] who reported two proved cases of the disease and a third presumptive case in 1933, was the first to apply the name of Waterhouse-Friderichsen syndrome to this clinical entity. Since the disease was first described by Little and since the first proved case was reported by Andrews, it would seem that if any eponym were to be employed—we personally are opposed to such terms—it should be called the Little-Andrews syndrome rather than the Waterhouse-Friderichsen syndrome, by which name it is now generally known.

Important reviews of the literature have been published by Aegerter[7] and Sacks[8] in 1937 and Kunstadter[9] in 1939. Aegerter's review included 57 cases, two of which he himself reported, and 55 of which had come to postmortem. According to Kunstadter, 73 cases are now on record, only 26 of which have been reported from the United States and only four of which, although the condition is most frequent in children, appear in the American pediatric literature. The case he reports brings the number in the latter group to five.

The exact number of cases on record is somewhat difficult to determine because of apparent duplications in the early literature and because of the inclusion, in some reports, of an occasional unproved case. It is interesting to observe, however, that the number of proved cases is still very small. Of the 38 cases collected by Bamatter[10] in 1934 the etiologic agent had been isolated in only 10, perhaps, as has been suggested, because of faulty methods em-

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*Read before the sixty-first annual meeting of the Louisiana State Medical Society, New Orleans, April 23, 1940.

†From the Departments of Pathology and Bacteriology of the School of Medicine of Louisiana State University and Charity Hospital of Louisiana at New Orleans.

ployed in many of the early cases. Since that time the meningococcus has been identified by Snelling and Erb[11] in three cases, by Aegerter in two, and by Boone and Hall,[12] Foucar,[9] Sacks, Sulzer,[9] DeBruin,[9] Weingart,[9] and Carey [13] in one case each.

The case reported by Carey represents, so far as we are able to determine, the single instance on record of recovery in this disease. In most of the reported cases death occurred within 24 hours or within an even shorter period of time. In the occasional case the duration was 48 hours. Carey's patient was a 27 year old white woman who presented a characteristic clinical picture of fulminating meningococcal septicemia with a purpuric rash. Cultures of the blood and the cerebrospinal fluid were positive for meningococci. Therapy, as suggested by Aegerter, consisted of cortical adrenal extract, infusions of dextrose and sodium chloride solution, and epinephrine. Antimeningococcic serum and sulfanilamide were also used, and the multiple therapy, as the author points out, makes it impossible to estimate the effectiveness of the attempted specific treatment with cortical adrenal extract. This is one of the very few cases on record in which the diagnosis was made antemortem and the patient lived long enough to permit specific therapeusis.

The following two cases are presented as classic clinical examples of this syndrome and also as illustrations of the comparative pathologic findings in cases of short and of relatively long duration.

CASE NO. 1

A white male child, 18 months of age, who had previously been perfectly well, was suddenly seized with convulsions at 4:00 p. m. March 11, 1938. He was admitted to Charity Hospital of Louisiana at 7:35 p. m. and died five minutes later, three hours and forty minutes after the first symptom had occurred. Stupor had alternated with convulsions prior to his admission to the hospital. The rectal temperature was 105° F., and the other positive findings included marked trismus, marked cyanosis, and a generalized purpuric rash. The time at which the purpura had appeared could not be determined. There was no rigidity of the neck.

Necropsy: Postmortem examination, which was performed two hours after death, revealed a well developed and well nourished male child who presented a generalized, mottled cyanosis of the skin not unlike postmortem lividity. A macular purpura was present and was most prominent over the face, neck, and upper thorax.

The serosal membranes of the pericardial, pleural and peritoneal cavities showed petechiae. The thymus, which weighed 30 gm., appeared normal. The heart, lungs, pancreas and gastrointestinal tract were congested but otherwise appeared normal. The mesenteric lymph modes were enlarged, and were soft and gray. The spleen weighed 45 gm. Its capsule was tense and the surface exposed by section showed a deeply congested, moderately firm pulp and prominent malpighian corpuscles. The liver weighed 460 gm. Its cut surface had a mottled yellowish-brown appearance.

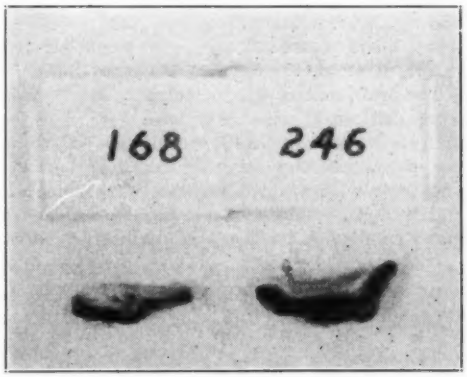

Fig. 1. Photographs of suprarenal glands of Case 1 (246) and Case 2 (168). Note the maintenance of the normal contour of each gland and the diffuse hemorrhage throughout the parenchyma.

Each adrenal (fig. 1) weighed 10 gm., in contrast to the normal average weight of 4.5 gm. The normal markings of each gland were masked by a diffuse, generalized hemorrhage, although the general contour was preserved. The fatty tissue surrounding each gland, as well as the perirenal fat, was edematous. The right kidney weighed 30 gm. and the left 47 gm. Both were markedly congested. The bladder and genital organs were normal.

The brain weighed 1,220 gm. Moderate edema was present, as evidenced by flattening of the convolutions. The meningeal vessels were intensely congested, but no hemorrhages were observed. There was no evidence of exudate in the subarachnoid spaces over the cerebral hemispheres or over the base of the brain. The hypophysis appeared normal.

The middle ears showed no exudate. The paranasal sinuses were not examined. Examination of the coronal sections of the brain after fixation revealed nothing additional.

Microscopic Examination: The sections were stained by hematoxylin and eosin. Mild parenchymatous degeneration and marked congestion were present in all the viscera. The pulmonary alveoli contained a considerable amount of fluid, but no cellular exudate. The liver showed a moderate degree of fatty metamorphosis. The germinal centers of the malpighian corpuscles showed swollen cells, irregular masses of a pink-staining coagulum, and a few polymorphonuclear leukocytes in the central portions. Diffuse hemorrhage, without inflammatory cell infiltration, was present throughout the medulla and cortex of the suprarenal glands. The few cells which could be recognized were swollen and irregular in outline. There was no site of predilection for the hemorrhage.

The skin showed marked congestion of the vessels of the corium, slightly swollen endothelial cells, and small accumulations of red blood cells around many blood vessels. There was no inflammatory cell infiltration.

The brain matrix was vacuolated. All the vessels were distended, some only with red blood cells, many with red blood cells and an appreciable number of marginating polymorphonuclear leukocytes, and some with a small number of red blood cells and a large number of polymorphonuclear cells. Many capillaries contained an acellular hyaline coagulum resembling hyaline thrombi. The nerve cells showed slight condensation of their cytoplasmic granules, and some of the glial cells had definitely granular nuclei. A few perivascular polymorphonuclear leukocytes were noted in the tissues of the pia in one small area over the cerebral cortex, but no exudate was present in the subarachnoid space. The veins, capillaries and blood sinuses of the choroid plexus were distended with blood, which showed many marginated polymorphonuclear leukocytes, a moderate number of which were present in the walls of some of the veins and sinuses and in the surrounding supporting tissue (fig. 2). In one area a few polymorphonuclear leukocytes and mononuclear cells were present on the ventricular surface of the epithelium, and occasional polymorphonuclear leukocytes were observed at the bases of the epithelial cells.

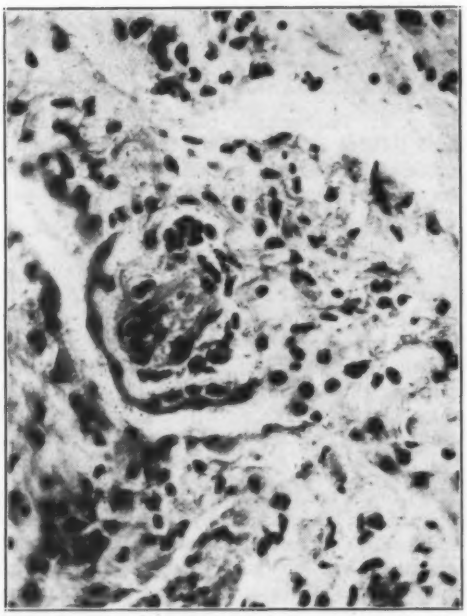

Fig. 2. Photomicrograph (x540) of choroid plexus of Case 1. Note the inflammatory cells in the lumen and around the vessel, which is located directly beneath the choroidal epithelium. Note also the moderate diffuse cellular infiltration in the supporting tissues.

Bacteriologic Studies: Brain broth (25 c. c.) was inoculated with blood from the heart (5 c. c.). After five days of incubation at 37°C., evidences of growth were observed, and a smear revealed gram-negative, biscuit-shaped diplococci of various sizes. No growth appeared on plain media, but excellent growths were obtained upon ascitic fluid blood agar. The organisms were morphologically and tinctorially typical meningococci, but it was only after repeated transfers on artificial media that agglutination by type II serum was finally accomplished.

CASE NO. 2

A white female child, 18 months of age, who had previously been perfectly healthy, suddenly became dyspneic at 8:00 p. m. February 7, 1940. She was intensely cyanotic for 15 to 20 minutes, and a rash rapidly appeared over the face and neck. She vomited several times, and during the night passed five loose stools, the last of which contained bright red blood.

She was admitted to Charity Hospital of Louisiana at New Orleans at 5:00 a. m. February 8 (nine hours after the onset). At this time the rectal temperature was 107°F. Physical examination revealed a well developed, well nourished child who was comatose and extremely dyspneic. A generalized macular purpura was most marked over the face and neck. The lips were cyanotic. Petechiae were present in the pharyngeal and buccal mucosae. There was no rigidity of the neck and the reflexes were normal. Lumbar puncture revealed clear spinal fluid, with no increase in cells or protein; no organisms were noted on direct smear.

Meningococcal septicemia was suspected, and 40,000 units of antimeningococcal serum were administered intravenously, and 10,000 units intrathecally. The child died at 8:45 a. m. February 8, 12 hours and 45 minutes after the onset of the first symptom. She vomited dark brown, bloody fluid shortly before death.

Necropsy: Postmortem examination, which was performed three hours after death, revealed a well

developed, well nourished white female child, who presented a generalized macular purpura, most marked over the face, neck and upper thorax (fig. 3). The entire body was involved except for a few small areas on the trunk and lower extremities.

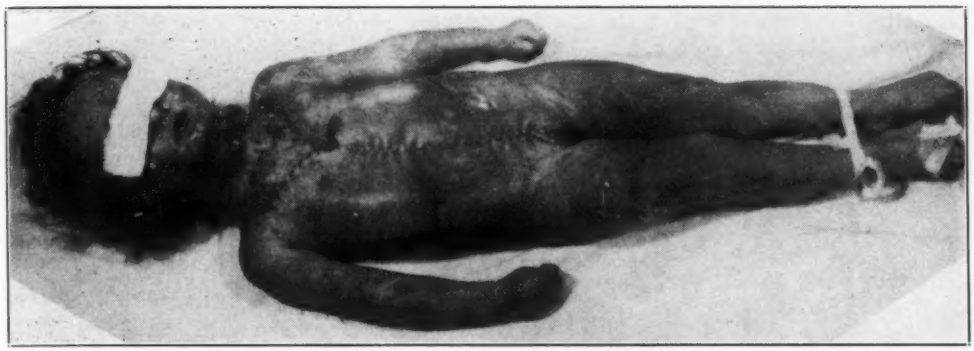

Fig. 3. Postmortem photograph of Case 2. Note the generalized discoloration and the macular purpura, which is most marked over the face, neck and upper thorax.

Straw-colored fluid (30 c. c.) was present in the pericardial cavity, and petechiae were observed in the pleural cavities. The peritoneal cavity was normal. The thymus, which weighed 22 gm., appeared normal. The musculature of the heart was flabby, but the organ was otherwise normal. The lungs were normal except for scattered petechiae. The spleen weighed 35 gm. Its capsule was tense and the cut surface revealed deeply congested, moderately firm pulp and prominent malpighian corpuscles. The liver weighed 400 gm. The parenchyma was soft, and the surface, on section, had a mottled, reddish-yellow color. No lesions were present in the pancreas. The mucosa of the gastrointestinal tract was deeply congested; the lymph follicles and Peyer's patches were prominent, and the mesenteric lymph nodes were enlarged and deeply congested.

Each adrenal gland was about twice the normal size. The normal markings were obliterated by diffuse hemorrhage, but the contour of each gland was well preserved (fig. 1). The genito-urinary organs were normal except for congestion.

The brain weighed 1,240 gm. The meningeal vessels were intensely congested and the brain substance was somewhat edematous. No exudate was noted in the subarachnoid space. No additional findings were observed on coronal sections. The middle ears were free of exudate. The superior surface of the cribiform plate showed hemorrhagic discoloration. The ethmoid cells contained a considerable amount of reddish-brown fluid, but the other paranasal sinuses were free from exudate. The hypophysis appeared normal.

Microscopic Examination: The visceral changes resembled those described in Case 1 except for the following points: The heart showed a slight degree and the liver a marked degree of fatty metamorphosis. The germinal centers of Peyer's patches, the mesenteric lymph nodes, and the malpighian corpuscles of the spleen were edematous. Many of the cells were swollen and necrotic, and phagocytosis of the granular nuclear debris was observed.

The hemorrhage into the suprarenal glands involved practically the whole organ, but was most prominent in the zona reticularis and the medulla. In some areas it was confined to the zona glomerulosa and the contiguous portion of the zona fasciculata, and the cells of the medulla were fairly well preserved in one small area.

The blood vessels in the corium of the skin showed congestion and perivascular hemorrhage. Frequently they contained fibrinous thrombi and endothelial cells which were swollen and vacuolated. An increased number of endothelial cells was seen in some of the blood vessels and lymphatics. Occasionally the lumen of a lymphatic vessel was filled with a fine network of fibrin.

A small area of rather extensive infiltration by polymorphonuclear leukocytes was observed in the tissues of the leptomeninx over the pons (fig. 4), though none were free in the subarachnoid space. The epithelial cells of the choroid plexus were somewhat hydropic. An occasional vessel showed margination of polymorphonuclear leukocytes, and an occasional cell of the same type was observed in the vessel wall and the supporting tissue.

Bacteriologic Studies: Direct smears made from material from the ethmoid cells beneath the cribiform plate, the right and left frontal sinuses, the

Fig. 4. Photomicrograph (x540) of the leptomeninx over the pons of Case 2. Note that the subarachnoid space is free of exudate and that the polymorphonuclear leukocytic infiltration is confined to the tissues.

purpuric spots, and the blood revealed gram-negative, biscuit-shaped diplococci of various sizes. A gram-negative diplococcus was observed on the hydrocele fluid agar slant inoculated with material from the paranasal sinuses but could not be isolated in pure culture. The same organism appeared in brain broth inoculated with heart blood. A feeble growth of gram-negative diplococci was noted on hydrocele fluid agar inoculated with this broth, but they were not viable after the first transfer. The growth was oxidase positive.

COMMENT

The two cases reported herewith correspond in most respects to the majority of reported cases, but show certain points of difference.

Both cases occurred in children under two years of age, which corresponds with the age incidence of most reported cases. According to Sacks, 70 per cent of the reported incidence is in children under this age. Attention has already been called to the fact that the first proved case occurred in an individual 53 years of age, and several other reported cases occurred in adults.

The symptoms and signs observed in these patients were observed in most of the reported cases. Headache, chills, dizziness and joint pains were also observed in some of the reported cases, chiefly in adults or older children in whom, with fever, they frequently constituted the initial symptoms. The initial symptoms in our own cases were convulsions and dyspnea, respectively. In most reported cases in infants and young children, however, the illness is likely to be initiated by a cry, followed closely by alternating cyanosis and pallor. Purpura usually appears in from three to 12 hours; it was absent in 13 of the 57 cases collected by Aegerter. In some of the reported cases a mild upper respiratory infection preceded the illness, and in Kunstadter's case otitis media was regarded as the etiologic factor.

All authors comment upon the dramatic speed with which symptoms and signs develop. In Kunstadter's case a graduate nurse observed the child (a 12 day old baby) become cyanotic and the skin become ecchymotic while she "watched dumfounded." When the physician saw the patient 15 minutes after the first symptom or sign was observed, ecchymotic areas, many very large, and petechiae covered the whole body.

It was possible in the second case which we are reporting to demonstrate organisms in direct smears from the blood and the purpuric spots, and this might have been possible in the first case had the proper examinations been made. Organisms were observed in many of the reported cases in which these examinations were made, and emphasis should be placed upon the importance of this procedure for early presumptive diagnosis, before blood cultures can be reported.

Although the clinical absence of meningitis in both these cases corresponds with similar findings in most of the reported cases, some microscopic evidence of meningeal inflammation was observed in both. In the first case there was moderate choroiditis and slight, localized, meningeal perivascular inflammatory cell infiltration. In the second case the choroiditis was slight and the meningeal infiltration more marked. A search of the literature has failed to reveal any reported case in which

choroiditis was observed. In the few instances in which meningitis was reported, the duration of symptoms before death was longer than in either of our cases.

It is interesting to observe that the patient who had hemorrhage over the cribiform plate and a hemorrhagic exudate in the ethmoid sinus had the greater involvement of the meninges (Case 2), whereas the patient with definite choroiditis had little meningeal cellular infiltration (Case 1). In Case 2 the involvement was localized to the base of the brain. The superior surfaces of the hemispheres appeared normal.

The characteristics of the hemorrhages into the suprarenal glands in both these cases corresponded with the characteristics of the hemorrhages in most of the other reported cases. The contour of the gland was maintained, there was an absence of inflammatory cellular infiltration, and the hemorrhage, although no part of the gland escaped, seemed to have a predilection for the medulla and zona reticularis.

In all the reported cases in which definite statements on this point were made, there was a marked hyperplasia of the lymph elements. In the first of our cases marked degenerative changes were noted in the germinal centers; in the second case the changes had gone on to necrosis, with phagocytosis of the cellular fragments. These findings are commonly observed in any severe toxic state and are not peculiar to the condition under discussion. It is our own opinion that the enlargement of the lymphoid structures observed during the first 12 hours of illness represents, for the most part, edema and necrosis rather than true hyperplasia.

Microscopic examination revealed only slight changes in the skin of the patient who lived less than four hours (Case 1), in contrast to swelling of the endothelial cells and the presence of thrombi in the blood and lymphatic vessels in the skin of the patient who lived 12 hours (Case 2). These findings seem to indicate that the injurious agent is probably directly responsible for the lesions, but that in many cases the disease is terminated by death before an inflammatory response beyond the vascular phenomenon can occur.

SUMMARY

Two additional cases of fulminating meningococcal septicemia are reported, in both of which microscopic evidences of meningeal inflammation were demonstrated, though there was no clinical evidence of it. Meningococci were cultured from the heart blood in both cases, and the same organisms were found in direct smears from the blood and the purpuric spots in the second case.

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DISCUSSION

Dr. Edwin H. Lawson (New Orleans): As the discussion of a paper so complete and so well presented as this essay of Drs. Moss and Schenken with the idea of presenting any additional information is almost impossible, it appears to be more appropriate at this time to stress certain points outlined in the paper.

Dr. Schenken has presented two cases of meningococcal meningitis which illustrate two of the four possible routes of transmission of the meningococcus from the nasopharyngeal mucosa to the meninges. The first illustrated the blood-borne type of infection in which the bacteria enter the blood stream and are transported by this means to the meninges. This Dr. Schenken has proved by the demonstration of the organism and the resulting inflammatory process in the choroid plexus. The second case illustrates the transmission of the

organism by way of the cribiform plate as demonstrated by Dr. Schenken in the sections removed from the cribiform plate which show the acute inflammatory process characteristic of such infection and the extension from this area to the meninges. The remaining two routes of extension are: (1) by way of the perilymphatic space around the olfactory nerve, and (2) by way of the ethmoid sinus. Although the routes mentioned above agree with the opinion of the majority of investigators along this line, some authorities still ascribe the route of extension as by way of the blood stream only.

Another point brought out by Dr. Schenken which should be emphasized is the fact that the diagnosis of meningococcal septicemia may be made in some instances by the examination of the blood smear for the presence of micro-organisms, inasmuch as in some cases of this disease the micro-organisms are so numerous that they may be demonstrated in a blood smear by use of an ordinary stain. The micro-organism may also be demonstrated in the material obtained from the skin lesions which occur in some cases of fulminating meningococcal septicemia.

The authors have mentioned the appearance of hemorrhage in the adrenal glands. The cause of such hemorrhage has been determined to be either an embolus of micro-organisms which might have located in a particular point in the capillaries of this organ or to the increased fragility of the capillaries causing a rupture at that point. In 1925 Dr. Loeber and I presented a case of hemorrhage into the adrenals of a newly born infant. In this particular case there was no evidence of septicemia, so that the cause of rupture of the capillaries was considered to be due to increased fragility of those blood vessels. The differentiation of these two particular features of this disease is still an unsettled question.

Dr. John H. Musser (New Orleans): One of the characteristic features of acute fulminating meningitis is the occurrence of purpura. Almost invariably, the children who go into the contagious disease division of Charity Hospital, and who die very promptly as a result of meningitis, all have severe purpura. Irrespective of what causes the purpura, in any acute infectious disease this is always a grave symptom. Furthermore, it seems to me that possibly in these two cases of Dr. Schenken, it was coincidence rather than anything else, that the purpuric lesions or the hemorrhage should happen to occur in the adrenal. As a matter of fact, as Dr. Schenken says, hemorrhages were not only located in the skin, but may have been here, there, and everywhere, in the parenchymatous organs. So, it is not quite clear whether it was coincidence, or whether it was some other factor, which was responsible for the involvement of the adrenals.

Dr. John R. Schenken (In conclusion): I did not think I had time to show the lantern slides. If I have time, I have only four I would like to show. (Showing slides) This is a photograph of the skin showing the diffuse cyanotic appearance of the skin, in addition to the purpuric lesions, most marked over the face, neck, and upper thorax.

This slide shows the suprarenal glands from both cases. Note the diffuse hemorrhage throughout.

This slide shows the meninges from the second case; note the definite cellular infiltration into the tissues, but none in the subarachnoid space.

The next slide shows the choroid plexus. Note the infiltration of polymorphonuclear leukocytes beneath the epithelium.

I am perfectly in agreement with Dr. Lawson that the cause of the hemorrhage in the skin may be an embolic phenomenon, but if I should venture an opinion on it, it seems to me, because of the definite evidence of injury to the endothelial cells that we see, that it is equally likely to be due to a local toxic injury to the endothelium, allowing for diapedesis of the red blood cells. I do not know what part a change in the fragility of the red cells, or a change in the bone marrow as far as the reduction in the number of platelets, may play.

Relative to the coincidence of hemorrhage in the suprarenals in these cases, I do not think I can answer Dr. Musser's question. It seems rather strange to me that the suprarenals should be picked out as the one organ in which considerable hemorrhage might occur. I grant also that the organ is rather small, but at the same time, unless we have been missing it routinely, organs of similar size, such as the hypophysis, should occasionally be involved as well as the suprarenals. But I do believe that the destruction of the suprarenals does add to the toxemia the effects of sudden acute suprarenal insufficiency.

THE MORTALITY OF ACUTE APPENDICITIS

A CONTINUING STUDY (4,207 CASES) FROM THE CHARITY HOSPITAL OF LOUISIANA AT NEW ORLEANS*

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The present contribution is the eighth of a series which I have prepared personally, or in the preparation of which I have done the chief work, concerning acute appendicitis at Charity Hospital of Louisi-

*Read before the sixty-first annual meeting of the Louisiana State Medical Society, New Orleans, April 23, 1940.

ana at New Orleans. The last of these papers, which was published in 1936, dealt with 2,715 surgical cases, and the present contribution brings to 4,207 the number of cases thus analyzed over the nine-year period ending April 1, 1939.

I think no apology is needed for the presentation of another study of this sort. The statistical method may be crude, the hospital records may be inaccurate and inadequate, and it may not always be easy to evaluate fairly material for which one is not oneself entirely responsible. But when all is said and done, the fact remains that the best method of studying acute appendicitis is by the determination of the case fatality.

Certain other points should also be stressed. For one thing, from the time this study was undertaken, in 1933, the repeated analyses have been made by the same persons, and whatever the factor of error due to the personal equation may be, it has at least been kept constant throughout. For another thing, the true mortality of acute appendicitis is the general hospital mortality, not the mortality of individual surgeons, which does not in any sense portray the true status of this disease. Finally, the mortality of this large public institution introduces no factor of exaggeration. A few years ago, when acute appendicitis was studied in the various hospitals of New Orleans, the mortality at Charity Hospital was found to be actually lower than the mortalities at several private hospitals.

The analysis with which I have occupied myself for the last several years concerns only acute appendicitis. It does not include any patient in whom, even though an emergency operation was performed, the disease proved to be not acute. I might say in passing that there should be no condemnation but only praise for surgeons who honestly err in operating for supposed acute disease. Their death rate will be considerably lower than the death rate of the surgeons who defer operation until the diagnosis of the disease is established—frequently by the development of gangrene, rupture and peritonitis.

More important, this series includes no chronic and no recurrent cases. Without entering into the question of whether or not chronic disease exists, such cases have nothing to do with acute appendicitis. For years we have been making the point that their inclusion in the statistics of the acute disease gives an entirely false idea of the situation, and is at least partially responsible for the complacency with which both lay and professional persons are prone to regard acute appendicitis. It is surprising to find how often surgeons who should and do know better are following this practice. I was happy to observe that the eminent statistician, Frederick L. Hoffman, in commenting upon our last report, took exactly the position that we did: Acute appendicitis is a public health problem, and the statistics of the acute and chronic varieties should never under any circumstances be discussed together.

MORTALITY ACCORDING TO PATHOLOGIC TYPES

In the 2,715 cases of acute appendicitis which we had accumulated for the six year period ending April 1, 1936, there were 157 deaths, 5.8 per cent. In the 1,492 cases which occurred in the three year period ending April 1, 1939, there were 79 deaths, 5.3 per cent. Even a fractional improvement in mortality in so common a disease as acute appendicitis results in the saving of a number of individual lives, but from the broader aspects so slight an improvement over a three year period is very discouraging. The mortality for the whole group of 4,207 cases was 236, 5.6 per cent.

A comparative study of the two series reveals some rather puzzling facts. The proportion of acute and suppurative cases in the combined series was almost 45 per cent of the total (1,890 of 4,207 cases). Since the chief method of reducing the mortality of acute appendicitis lies in increasing the proportion of cases in this particular group, it was gratifying to find that the proportion of 41.5 per cent in the first series (1,137 of 2,715 cases) had risen to 50.4 per cent in the second (753 of 1,492 cases). It was disturbing, however, to

find that the mortality of 0.34 per cent for this group in the first series (five cases) had risen to 1.59 per cent (12 cases) in the second series, more than four times as high.

It is not utopian to conceive of a day in which gangrene and rupture will be considered complications of acute appendicitis rather than integral parts of the disease, but for the present they must be discussed on the latter basis. In the whole group and in the first and second series the proportion of cases of gangrenous appendicitis was practically the same, just over a quarter of the total (1,096 of 4,207 cases, 711 of 2,715 cases, and 385 of 1,492 cases respectively). The mortality for the whole group was 5.47 per cent (60 cases), but again there was a disturbing rise from the mortality of 3.1 per cent in the first series (22 cases) to 9.87 per cent in the second series (38 cases).

Undoubtedly a portion of the mortality which in the first series was due to rupture in the second series was due to gangrene, for in the second series there was a striking decrease in both the incidence and the mortality of ruptured appendicitis. The proportion of ruptured cases in the combined series was 21.3 per cent (989 of 4,207 cases) and the combined mortality was 13.8 per cent (124 cases). In the first series rupture represented 23 per cent of the cases (615 of 2,715 cases) and the mortality, because of the fact that more than a third (244 cases) were associated with generalized peritonitis, was 17.2 per cent (112 cases). In the second series the proportion of ruptured cases had fallen to 19 per cent (283 of 1,492 cases) and the mortality had fallen to 4.17 per cent (12 cases), apparently because less than 20 per cent (59 cases) were associated with generalized peritonitis.

Although the decrease in the incidence of generalized peritonitis is undoubtedly the most hopeful trend in the second series of cases, it cannot be accepted entirely at its face value. We have studied only the surgical cases of acute appendicitis, it is true, but we have examined the non-surgical cases treated conservatively, and we have studied critically every non-surgical death which occurred in the entire period of the analysis. Clearly part of the reduction in the mortality of ruptured appendicitis in the second series is due to a more judicious use of conservative therapy in selected cases. On the other hand, it is unfortunately impossible to escape the conclusion that part of the increase in the number of non-surgical deaths, which were roughly the same in the second three year period (50 cases) as in the first six year period (51 cases), was due to an injudicious use of this type of therapy.

It would be both unfair and foolish to say that certain patients might have lived if they had been operated on rather than treated conservatively. It can be said in all fairness, however, that the second group of deaths seems to include a larger number of cases than the first group in which, on the surface, conservative therapy seems to have been employed on somewhat doubtful indications.

Furthermore, the figures for appendiceal abscess, in addition to raising the question as to whether this termination is a consummation so devoutly to be wished as some of the recent literature would seem to suggest, also raises the question as to whether conservative therapy may not, perhaps, be in danger of being carried too far. Abscess occurred in 7.6 per cent of all cases in the combined groups (323 of 4,207 cases) and was attended with a mortality of 10.8 per cent (35 cases). It represented 9.3 per cent of the cases in the first series (252 of 2,715 cases) and was attended with a mortality of 7.1 per cent (18 cases). In the second series the incidence had fallen to 4.7 per cent (71 of 1,492 cases) but the mortality had risen to 23.9 per cent (17 cases). Furthermore, in the second figures are included four cases of cul de sac abscess, another outcome usually regarded as favorable, which were treated by colpotomy or proctotomy, with a mortality of precisely 100 per cent.

In view of the implied criticism of conservative therapy just expressed, it might be well to make clear my views concerning

it. I regard it as an extremely valuable method in selected cases of ruptured appendicitis with peritonitis if it is used, as an English surgeon expressed it, "on the threshold of the operating room." I think I would add to that precaution, if it is also used with one's heart in one's mouth. It is not the treatment of the early case of acute appendicitis, though in certain fatal non-surgical cases which I examined it was employed as early as 24 hours after the onset of symptoms. A very fair proportion of the fatal non-surgical group, furthermore, was made up of young children, individuals advanced in years, and negroes, in all of whom I believe this method should be used with the greatest caution, if at all. The same holds for its use in pregnant women. Finally, in spite of the extreme value of this method in selected cases, I share the view expressed by Ochsner and others, that immediate operation in all cases of acute appendicitis, regardless of when the patient is seen or what complications may be present, will result in fewer fatalities in the hands of the general run of surgeons than will the practice of conservative therapy by men who do not thoroughly understand its limitations and implications.

MORTALITY ACCORDING TO AGE GROUPS

It is scarcely necessary to labor the point that the mortality of acute appendicitis with and without peritonitis is as far apart as the poles. For the combined series, exclusive of abscess cases, the mortality without peritonitis was 1.7 per cent (53 of 3,105 cases) and with peritonitis was 25.7 per cent (148 of 575 cases). The mortality for individuals under 12 and for individuals between 13 and 39 was practically the same without peritonitis, slightly over 1 per cent, and with peritonitis, slightly over 24 per cent. In the group over 40 years of age, however, the mortality without peritonitis was 8.2 per cent (22 of 252 cases) and with peritonitis was 31.2 per cent (38 of 119 cases). The striking feature of this special analysis, aside from the shocking mortality in individuals advanced in years, is that peritonitis in the middle years was present in less than 10 per cent of all cases (262 of 3,048), whereas it was present in more than 25 per cent of the cases under 12 years of age (194 of 738 cases) and in almost 30 per cent (119 of 421 cases) in individuals over 40 years of age.

Both the high incidence of peritonitis and the high death rate furnish proof of the extreme seriousness of acute appendicitis at the extremes of life. The collected figures for both series show that the incidence of the disease below 12 and over 40 years of age was just over a quarter of the total cases (27.5 per cent, 738 children, 421 adults over 40 years of age), whereas the deaths in these age groups represented considerably more than half of the total mortality (53.9 per cent, 62 children, 65 adults over 40 years of age). There was some improvement in the death rate in children under 12 years of age in the second series (7.1 against 9.0 per cent), but the improvement in the middle years was merely fractional (3.5 against 3.6 per cent), and the mortality in the group over 40 years of age had actually risen in the second series, albeit fractionally (15.9 against 15.2 per cent).

MORTALITY IN RELATION TO PURGATION

The habit of purgation persisted in the second series, although for some reason the mortality was considerably lower in connection with it (27 of 418 cases, 6.4 per cent, as compared with 76 of 795 cases, 9.5 per cent). The mortality of repeated purgation in the combined series was 16.2 per cent (42 of 260 cases). Physicians, regrettably, are still ordering purgatives for abdominal pain, and to that habit can be attributed a mortality of 26 per cent (13 of 50 cases) in the combined series. The second series, moreover, contained the happily unique record of a physician who diagnosed the patient's trouble as acute appendicitis and forthwith ordered a dose of salts.

On the other hand, it was encouraging to note in this series the increasing number of physicians who warned their patients against food and purgation and sent them into the hospital immediately the diagnosis was made or suspected. This trend in the

new series is both outstanding and hopeful. Some mention should certainly be made, also, of the single druggist who acquired merit by doing precisely the same thing.

MORTALITY ACCORDING TO RACE

The mortality for white patients in the combined series was 3.36 per cent (142 of 3,245 cases) and for negroes 9.77 per cent (94 of 962 cases), the improvement for both races in the second series being merely fractional. Negroes, it will be noted, furnished less than a quarter of the total cases (22.8 per cent) but well over a third of the mortality (39.8 per cent).

Some explanation for the lack of improvement in the negro mortality lies in the fact that in the second series 38 per cent of the negro patients (135 of 356) took purgatives, against only 25 per cent (285 of 1,136) of the white patients. Another explanation lies in the fact that almost half of the white patients (47.1 per cent) were admitted to the hospital within the first 24 hours of illness, against just over a third of the negro patients (34.4 per cent). These comparative figures explain why roughly 62 per cent of all cases of appendicitis in the negro had passed beyond the simple acute or acute suppurative stage when the patients were first seen, as compared with 45.6 per cent in the white patients. There is also no doubt in my mind, regardless of these explanations, that acute appendicitis is a more severe disease in the negro, perhaps because it is acquired.

MORTALITY ACCORDING TO OPERATIVE PROCEDURE

The mortality of simple appendectomy was practically the same in both series, 1.3 and 1.4 per cent respectively. The mortality for other operations, however, varied widely, in correspondence with the differences in types of pathology and the resulting mortalities, upon which I have already commented. The mortality for appendectomy and drainage in the first series was 6.0 per cent (17 of 284 cases) but for the second had risen to 13.4 per cent (31 of 231 cases). The mortality of incision and drainage in the first series was 13.9 per cent (19 of 137 cases) but for the second had risen to 27.5 per cent (16 of 56 cases).

Enterostomy in the second series was apparently reserved for the most seriously ill patients, for it was performed only 29 times, with a mortality of 38.6 per cent (11 cases), as compared with 371 times in the first series, with a mortality of 22.6 per cent (84 cases). Whether the mortality in the second series would have been lowered by a more extensive use of this auxiliary procedure it is not possible to say.

In the combined series 20 cases were treated by colpotomy or proctotomy alone, with 10 deaths, a mortality of 50 per cent. Such statistics, it must be said again, do not encourage us to regard the formation of cul de sac abscesses as the solution of the problem of ruptured appendicitis with generalized peritonitis.

OTHER CONSIDERATIONS

It is not possible, in such a contribution as this, further to analyze statistically the data in these cases, but certain general statements can be made. The postoperative care of appendiceal cases at Charity Hospital has usually been very good, and the addition of constant suction or intestinal decompression to the regimen, as well as to the regimen of conservative therapy, was a striking improvement in the second series. Perfringens serum and sulfanilamide therapy were used too infrequently to permit conclusions. Bower's work with the former, and the various reports already in the literature concerning the latter, suggest that later reports from Charity Hospital will show a wider and more profitable use of these measures.

A survey of the records makes it very clear that he is a bold man, and not a very prudent man, and, if he is a teacher or writer, a rather dangerous man who generalizes about acute appendicitis. The average leukocytosis, for instance, may be between 10,000 and 15,000, but less than one-third of these cases fell into that group, and the range was from 3,200 to 43,200.

Fever was listed as an invariable accompaniment of acute appendicitis by the great John B. Murphy, who seems not to have escaped the trap of generalizations. But 883 of the patients in this series, nearly 21

per cent of the total, had no fever on admission, and almost exactly the same number (884) had pulse rates below 80 on admission. The 21 deaths in the fever-free group, and the 15 deaths in the group with normal pulse rates, strongly suggest that some physicians, at least, were unwilling to diagnose acute appendicitis until alterations had occurred in these respects.

As for symptoms, the only safe generalization is that pain is practically always present. In these cases it was the first symptom in by far the largest proportion of cases, and it occurred in all locations, including the back, the loin, the scrotum and the leg. Nausea and vomiting, although they were very frequent first symptoms, were not present at all in a fair number of cases. Other first symptoms included such atypical ones as diarrhea, urinary disturbances, headache, vertigo, malaise, and syncope. The onset occurred during sleep, at work, at all sorts of play, during the act of defecation, during menstruation, after tonsillectomy and after cataract extraction, after vaccination, after the extraction of teeth, and in the course of malaria, paratyphoid and typhoid fever, pneumonia, influenza, measles, and acute pelvic inflammatory disease.

The onset occurred following dietary indiscretions in a small but highly fatal group of cases. It followed previous incision and drainage of abscesses, or the previous conservative treatment of peritonitis, in another small group of cases, the death rate in which emphasizes the importance of urging prompt interval appendectomy upon patients who have been so treated. Finally, the small group of cases must be mentioned in which the taking of a purgative immediately preceded or was associated with the onset of symptoms, a situation obviously fraught with serious possibilities.

Bower's studies on the importance of the *Clostridium welchii* and Wangensteen's demonstration of the obstructive character of acute appendicitis are the only really significant contributions to the subject of acute appendicitis in recent years except for the work of Bower and his associates in Philadelphia. That is the most important contribution of all, for it shows what can be achieved by a continuing campaign of public education. For that matter, Hoffman, in his yearly analysis of the 1936 appendicitis death rate, attributed the decrease in New Orleans to the brief educational campaign conducted in that year, which I had the privilege of directing in the absence of the appointed chairman from the city during its active portion. Like the Philadelphia campaign, the New Orleans campaign concentrated on two points, abstinence from purgation in abdominal pain, and prompt operation in acute appendicitis, before rupture occurs.

Some special comment must be made concerning the unruptured appendix. Operation on the simple acute or acute suppurative appendix is the desideratum. Operation on the gangrenous appendix, even though it is intact, is not in the same category of safety. The figures from Charity Hospital make it abundantly clear that there is no warrant for taking a lighthearted view of gangrene in appendiceal disease, for the mortality in such cases is very far from negligible.

THE PRESENT STATUS OF APPENDICEAL DISEASE

I am not sure that I am willing to accept Bower's suggestion, regardless of the authority with which he speaks, that medical men cease to write and medical editors cease to accept articles on acute appendicitis, and that all our attention, instead, be concentrated on rupture and peritonitis. That peritonitis is the cause of most deaths in this disease there can be do doubt. But my own opinion is that one of the reasons for that situation is not that too little attention is paid to peritonitis but that too little is paid to acute appendicitis, *per se*. The paradox still exists that peritonitis develops because physicians fail to recognize the syndrome of uncomplicated acute appendicitis.

For that reason, Fitz's suggestion is timely, that acute appendicitis be taught in the courses in medicine as well as in the courses in surgery, on the ground that medical men rather than surgeons hold the

clue to the solution of the problem. In the same connection, I am inclined to suggest the scrapping of most of the descriptions of acute appendicitis in most of even the modern textbooks. If they do not actually give the impression that gangrene, rupture and peritonitis form part of the initial syndrome of the disease, they are so languid, so unemphatic, that the student gets no idea whatsoever of the urgency of acute appendicitis. I was enormously amused by the comment of Dr. J. D. Rives to the effect that the description of acute appendicitis in the textbook then in use in the L. S. U. Department of Surgery sounded like "something that might have happened to King Tut." The comment is less funny, however, when one recollects that from such descriptions medical students are getting their first introduction to the most frequent of all surgical diseases.

Loveland, writing in the Yale Journal of Biology and Medicine, said that Fitz in 1886 had told medical men nearly everything of importance they were to learn in the next half century, and even down to the present time, about acute appendicitis, and it is doubtful whether modern surgery has yet caught up with those ideas: immediate operation, no purgation, watchful waiting in neglected abscesses, and interval appendectomy. That surgery is not equal to the situation I doubt. The solution, I think, lies rather in the continuing and constant education of the public by the medical profession. Anyone who doubts that conclusion need only study the records of acute appendicitis in the Charity Hospital of Louisiana at New Orleans.

SUMMARY AND CONCLUSIONS

1. In this, the eighth of a series of studies on acute appendicitis at Charity Hospital of Louisiana at New Orleans, the number of cases analyzed for the nine year period ending April 1, 1939, has been brought up to 4,207.

2. A study of the total series, and a study of the last series as compared with the (cumulative) former series, show a practically stationary mortality, such improvement as has occurred in certain regards being offset by regression in other regards.

3. The mortality for the second series continued unduly high for children under 12 years of age, adults over 40 years of age, and negroes. The mortality was very high when purgatives were taken, and inordinately high when they were repeated.

4. Certain statistics in the second series introduce the question as to whether conservative treatment was used wisely in all cases.

5. A survey of the records from the standpoint of symptomatology, pulse rate, temperature, leukocytosis, mode of onset and similar factors makes it clear that generalizations on any of these subjects are extremely unwise.

6. The conclusion is inevitable that only by a continuing campaign of public education, based on prompt operation and abstinence from purgation in abdominal pain, can the mortality of acute appendicitis be reduced to a reasonable figure.

DISCUSSION

Dr. Oscar W. Bethea (New Orleans): I do not think that there is any doubt that one of the most impressive pieces of work done in New Orleans has been the study of appendicitis by Dr. Boyce and his associates during the last few years. Personally I would say they have largely revolutionized my ideas of this condition. The emphatic fact that they have demonstrated is the value in acute appendicitis of early operation. The surgeon is held responsible for unfavorable results, yet the surgeon is not to blame except in a fairly small percentage of cases. The patient is usually seen first by an internist, pediatrician, gastroenterologist, general practitioner or some one in one of the related groups. I can only contribute, I believe today, by making some apology for the medical groups that have the responsibility of the case in the first few hours. One of the greatest difficulties that we experience is making a tentative diagnosis so that the preliminary disposition of the patient can be made, and a surgical consultant seen. It has been said that medicine is part science, part skill and part salesmanship. One difficulty encountered in medicine is selling the patient and the patient's family the idea of going to the hospital. Even at the suggestion of possible appendicitis we are up against an antagonistic reaction on the part of the patient and his family in many instances. The doctor is told that he is accustomed to having these attacks. We are told that "Dr. Smith used to come in and give him a prescription

and he was well the next day." "Just last week a man working in the office had an attack just exactly like this and took a dose of salts and was back at work the next day." We have to sell them the idea of going to the hospital.

One thing I would like to emphasize is that medical students have been impressed with the idea that there are four symptoms; pain, tenderness, fever and nausea and, to a less extent, changes in the blood picture. Usually the patient has to be moved to the hospital before we can demonstrate the last. I would like to mention a point stressed by Dr. Coleman, that increase in pulse rate is definitely suggestive of a grave condition. I remember that he would refuse to take the responsibility of a case that was not immediately removed to a hospital if there was a marked increase in pulse rate. Another thing is temperature. This is not usually taken correctly. I use an accurate thermometer, and leave it under the tongue for five minutes and watch the lips of the patient during this time. If the patient talks he lets in cool air which prevents a correct reading.

I believe that the family physician, the internist, or whoever sees the patient first should share in the responsibility of the operation in those cases where there is an element of doubt; convincing the family that even though this may be a normal appendix, the patient requires surgical investigation.

In closing, I would say that I believe the internist may have a field of usefulness in deciding upon the anesthetic and the status of the surgical risk from the standpoint of the heart, lungs and vascular apparatus. The internist is also sometimes of service in watching the progress of some patients postoperatively especially when there is a circulatory or a respiratory handicap.

Dr. D. I. Hirsch (Monroe): One thing that impressed me was the remarkably low mortality in cases of acute appendicitis at the Charity Hospital in New Orleans. It is probably the lowest of any general hospital in the United States.

The one thing about appendicitis is this: Appendicitis is a surgical condition at all times. Another thing that tends to increase the mortality is the difficulty in diagnosis of appendicitis. It is the most common surgical condition we have and at times the most difficult to diagnose. I have observed cases of appendicitis with abscess formation in which the patients were not sick. In one case of abscess the boy had never been confined to his bed.

In order to appreciate the conditions that are taking place in the abdomen we should have a complete understanding of the pathology that exists. Of course we all know that when the appendix becomes gangrenous or ruptures the pain does cease for a while but there are so many angles to the diagnosis and the time is too short to discuss them. We can only speak of generalities.

Another thing I think should be mentioned is the use of opiates while attempting to make a diagnosis. Sometimes the pain is not so severe and we tend to relieve the patient with a small hypodermic and the first thing we know we find the patient has peritonitis. Surgery adds an infinitesimal amount to the mortality of appendicitis but delay is the serious thing.

In closing I would like to say that besides the educational features for the public, I think we members of the profession need to be educated too. I do not think there has been enough emphasis placed upon the seriousness and difficulty in diagnosing appendicitis.

I do not have time to discuss treatment. There is no question about immediate operation in acute appendicitis. Another thing, there is no doubt in my mind that surgery or improper technic or too much surgery at times will add to the mortality. There is a place in cases of appendicitis for conservative surgery. You can sometimes drain and if necessary go back later and remove the appendix. I think all persons with obscure abdominal pain which persists for six hours, regardless of temperature or laboratory findings, that cannot be reasonably accounted for should have the abdomen opened.

Dr. Alton Ochsner (New Orleans): I do not think that Dr. Boyce need be apologetic for bringing this subject to our attention. There are a few points which I should like to make. One is that in appendicitis the mortality rate in the two extremes of life, as he emphasized, probably is not entirely due to the lack of resistance in the young and elderly. One factor is that the diagnosis is more difficult in these cases.

I should like to say a word about conservative treatment. There is no such thing as conservative treatment of appendicitis. Dr. A. J. Oschner never practiced conservative treatment of appendicitis, but did use it in appendiceal peritonitis. There is only one treatment of appendicitis and that is removal of the appendix while the infection is still confined to it. The diagnosis of appendicitis is not always easy, however. At one time I was critical of myself if I operated upon a patient and found a normal appendix, but am no longer so. There is difficulty in making diagnosis. I think if a man is honest in his attempt to make a diagnosis in a suspected case and if he is reasonably certain by ruling out other lesions which might account for the symptoms and feels the patient has appendicitis, the patient should be operated upon. If one will contrast the mortality and morbidity of cases of ruptured appendicitis with the safety of appendectomy not associated with rupture, the justification of such a conclusion is evident. The important thing brought out today is insistence upon early operation.

In regard to therapy, advances have been made. One is the use of sulfanilamide in cases of ap-

pendiceal peritonitis, which we have used with good results. The mortality in these cases will decrease.

Another point is the use of adrenal cortical extract when the patients have severe toxemia. Sometimes adrenal cortical deficiency is responsible for death. It is surprising how much better these patients get along with the introduction of the extract.

THE MANAGEMENT OF THE THIRD STAGE OF LABOR*

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The rather general belief that the successful termination of the second stage of labor is equivalent to the successful termination of the entire process of parturition is by no means justified. As a matter of fact, more serious complications arise during the third stage than during the other two stages combined. Furthermore, a high percentage of these complications is due to mismanagement. The normal mechanism of the third stage is frequently disturbed by attempts to hasten its conclusion. Almost as frequently a persistent "hands-off" policy in the presence of abnormalities which demand intervention also results in catastrophe.

SEPARATION AND EXPRESSION OF THE PLACENTA

For the intelligent management of the third stage of labor, the immediate recognition of complete placental detachment is essential. Although no sign is infallible, when uteroplacental separation has occurred and the placenta has descended into the lower uterine segment, the process is likely to be manifested by the following signs: elevation of the fundus; bulging in the suprapubic region; flattening of the uterus; advancement of the cord; failure of the cord to retract on pressure over the suprapubic region; absence of fundal impulse in the cord; a slight spurting of blood.

Another important consideration is that the process of placental detachment takes place much more rapidly than we formerly believed. In a recent analysis of 1,000 consecutive cases of our own, it was found that in 62 per cent the duration of the third stage was less than five minutes. Other data concerning the rapidity of placental separation have been accumulated by clinical observation during cesarean section, by soft tissue roentgenology, and by hysterotokographic tracings.

Premature attempts to express the placenta before separation has occurred merely complicate the normal mechanism of detachment and should be avoided. Indeed, such procedures as kneading, squeezing and massaging the abdomen in an attempt to hasten placental separation are likely to result in partial detachment and more or less profuse bleeding.

Once the placenta is detached, however, it should be expressed at once if it is not expelled spontaneously. The detachment of the placenta is a natural mechanism, but delivery is very seldom a spontaneous process, particularly with the modern tendency to use analgesia or anesthesia in most cases. When spontaneous expulsion is impossible, some method of expression must be used. If the patient is conscious and cooperative, she may force the placenta out by her own efforts while the physician grasps the recti muscles in the midline at the umbilicus (indirect expression). If this method fails, the fundus of the uterus may be grasped firmly and the placenta delivered by gentle squeezing and downward pressure in the axis of the birth canal (simple expression). By the Credé technic, which should be used only if repeated attempts at simpler methods have failed, and which is applicable to both the attached and the detached placenta, the placenta is literally squeezed out of the uterus as if it were the pit of a cherry.

Certain precautions are necessary, whatever mode of expression is used. In the first place, the manipulation should not be un-

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dertaken until the uterus is firmly contracted. In the second place, too forceful downward pressure should be avoided. Finally, the uterus should be carried up out of the pelvis by suprapubic pressure after the placenta has been delivered, because uterine prolapse interferes with the normal mechanism of retraction and contraction.

Following its expression, the placenta should be carefully inspected to make certain that no part of it remains in the uterus, this being a simple precaution which will avert many anxieties in the puerperal period. Another reason for careful examination is to make certain that an accessory lobe or a placenta succenturiata is not present. Membranes which have been incompletely expressed may be ignored unless bleeding is profuse, but placental tissue must be removed immediately, to forestall hemorrhage and infection; secondary removal is an extremely hazardous procedure.

MANUAL REMOVAL OF THE PLACENTA

Manual removal of the placenta is occasionally necessary, but it is a formidable procedure at best and is the least desirable method of terminating the third stage of labor. Rigid standards should therefore be set up concerning both the indications for and the technic of the procedure.

Manual removal of the retained placenta should not be resorted to, in the absence of specific indications, until it is clear that simple methods of expression will not succeed. Credé expression under anesthesia is often effective if oxytocics are administered before the attempt is made. Even an hourglass contraction of the uterus often relaxes following the administration of an anesthetic or of adrenalin (minims 7), atropine (gr. 1/150), or nitroglycerine (gr. 1/100). On the other hand, it must not be forgotten that while these attempts are in process, serious hemorrhage may occur, usually due to partial separation of the placenta. Furthermore, violent manipulations are extremely dangerous, for they may traumatize the uterine tissues or even result in complete uterine inversion.

There are three major indications for manual removal of the placenta, namely, hemorrhage, retention of the placenta, and the desire or necessity for manual exploration following difficult delivery, in order to complete the labor without delay or to rule out uterine rupture. Often more than one of these reasons is effective.

Free bleeding is the most frequent indication for manual removal of the placenta, though all bleeding during the third stage of labor is not necessarily of placental origin. If the placenta is still firmly attached and if the uterus is firmly contracted, hemorrhage is usually due to lacerations of the parturient canal. If, on the other hand, the uterus is soft and boggy, the bleeding is probably due either to partial separation of the placenta or, less frequently, to its retention in the uterus after complete separation. Under these conditions the large sinuses of the placental site are exposed, and hemostasis does not occur because the mechanism of uterine retraction and contraction is unsatisfactory.

No exact criterion can be set up as to the amount of blood loss which furnishes the proper indication for placental extraction. Bleeding must not be allowed to continue indefinitely, however, and, generally speaking, a loss of 300 c. c., following the failure of attempts at expression of the placenta by simple methods, should be regarded as an indication for the more radical procedure.

When the placenta is not expelled either spontaneously or by some simple mechanical method soon after delivery, a brief period of delay is justified in the absence of free bleeding. If it is still *in situ* at the end of an hour, manual removal should be resorted to, for prolonged retention is not without risk. Deferred removal is often difficult and dangerous because of spastic conditions and contraction rings of the uterus, which are usually found several hours after delivery.

Arrest of the placenta within a tightly contracted cervix is naturally not included in this discussion, for it can readily be removed by the shoehorn maneuver without invasion of the uterine cavity. Furthermore, some distinction should be made between the attached and the detached re-

tained placenta. Conservative therapy is justified in certain cases of detachment. Our own experience has been that soft tissue roentgenology may be helpful in differentiating between detached and attached retained placentae, and we should be inclined to recommend that this diagnostic aid be employed routinely when it is at hand.

The patient with a retained placenta should never be left unattended, either in the hospital or the home, for fatal hemorrhage may occur in a very short period of time. In prolonged placental retention with infection, the best plan is probably to omit all attempts at manual extraction of the placenta and resort immediately to hysterectomy.

The wisdom of exploring the uterus, to rule out laceration or rupture following such traumatic procedures as a difficult forceps delivery, version and extraction, or craniotomy, is so obvious as to need no discussion.

A study of the obstetric records at Charity Hospital of Louisiana for the decade ending January 1, 1940, supports the point of view we have advanced concerning manual removal of the placenta. During the 10 year period the procedure was resorted to in 152 cases, one in every 250 viable deliveries. Free bleeding was an indication in 56 per cent of the group, retention of the placenta in 20 per cent, and exploration of the uterus following difficult labors or operative deliveries in 24 per cent. In four cases of the latter group rupture of the uterus was thus identified, and further proof of the wisdom of such exploration is found in the fact that during the same period of time uterine tears remained undiagnosed in seven fatal cases in which digital examination of the uterus was omitted. Death occurred in 10 of the 152 cases, 6.5 per cent, in which manual removal of the placenta was undertaken, a high death rate which is partially due to the basic pathology but which would be justified, regardless, by the fact that death also occurred in 13 cases during the decade studied in which a policy of non-intervention was practiced in the presence of placental retention.

Manual removal of the placenta, as has been pointed out, is a major obstetric operation. Asepsis must be meticulous, and it is well to change the gown, gloves and linen which have been used during delivery. In cases in which manual removal is likely to be necessary, it is best not to repair episiotomies and other lacerations prior to delivery of the placenta, for the repairs may be undone during subsequent manipulations.

Anesthesia is essential. One hand supports the uterus and forces it into the pelvis by pressure on the abdomen. The other hand, well lubricated, is introduced into the amniotic sac and follows the cord to the site of the insertion of the placenta. All manipulations are conducted within the sac, and the hand is not withdrawn until the entire operation is completed. The placenta is slowly peeled off and removed, together with the membranes, the tissues then being examined carefully to make certain that the removal has been complete. The use of ergotrate by the intravenous route usually obviates the necessity for uterine packing and thus lessens the possibilities of infection.

THE USE OF OXYTOCIC DRUGS IN THE THIRD STAGE OF LABOR

Before proceeding to the discussion of pathologic abnormalities of the third stage of labor, the use of oxytocic drugs must be briefly mentioned. Textbooks of obstetrics are vague and indecisive on this point, and widely divergent views seem to be held concerning their value. With the idea of securing a cross section of American obstetric opinion, a questionnaire was sent to a large group of representative obstetricians, many of whom control important clinical services and direct teaching departments of obstetrics in important medical schools (table I). The following discussion therefore represents not only our personal experiences but the experiences of a significant group of American obstetricians, 126 of whom replied to our questionnaire.

TABLE I

QUESTIONNAIRE ON USE OF OXYTOCICS
ADVOCATE ROUTINE OXYTOCIC

Yes—112 or 88% No—14 or 12%
ADMINISTRATION

Oxytocic	End of second stage No.	%	End of third stage No.	%	Both No.	%	Totals— No.	%
1. Ergonovine	21	46	22	48	3	6	46	36
2. Pituitary extract	28	70	10	25	2	5	40	32
3. 1 + 2	Pituitrin		Ergotrate		26	100	26	20
4. Pitocin	3	38	5	62	0	0	8	7
5. 1 + 4	Pitocin		Ergotrate		6	100	6	5
Totals	52	42	37	29	37	29	126	100

We have personally studied the efficacy of various oxytocics by comparisons of results achieved with them in small series of consecutive normal cases in which spontaneous expression of the placenta occurred (table II). Blood losses were determined by an exact chemical-colorimetric method whereby the amount of hemoglobin loss (in grams) in free blood, blood clots, and sponges is converted into cubic centimeters of blood. This is a more exact method of determining blood loss than either clinical estimation, which is usually inaccurate, or volumetric measurement of all fluid lost, which usually includes amniotic fluid and urine as well as blood. Our studies show clearly that the administration of an oxytocic at the end of the second stage of labor shortens the placental stage by half, decreases the blood loss by half, and markedly decreases the danger of postpartal hemorrhage. Our own experience is borne out by the opinion of the obstetricians whom we questioned: 88 per cent of the 126 advocate the routine use of an oxytocic drug at delivery, and 71 per cent prefer to use it at the end of the second stage.

TABLE II
EFFECTS OF OXYTOCIC THERAPY IN THIRD STAGE OF LABOR

	No. of cases	Averages— Blood loss—c.c.	Duration in minutes	Complications
No Oxytocic	50	212	8.4	One excessive bleeding
Pituitrin intramuscularly End of 3rd stage	25	198	7.1	One postpartal hemorrhage One excessive bleeding
Pituitrin intramuscularly End of 2nd stage	25	102	4.4	0
Pitocin intramuscularly End of 2nd stage	25	86	3.7	0
Ergotrate intravenously End of 2nd stage	25	54	2.1	One retained placenta
Totals	150	144	5.6	

Some form of ergonovine, usually ergotrate, in dosages of 0.2 mg. (gr. 1/320) was advocated by 61 per cent of the obstetricians questioned, either alone or in combination with other oxytocics. It has been our experience that this particular preparation is extremely effective. It acts within one minute when given intravenously and within two to four minutes when given intramuscularly, the tetanic uterine contractions which result often persisting for more than an hour without intervals of relaxation. It seems to have no detrimental effects on blood pressure, pulse, or urinary output. It has the additional advantage that it can be given by mouth if the patient is awake. We have found ergonovine of distinct value in both the prophylaxis and the treatment of postpartal hemorrhage due to atony, and by using it routinely we have materially reduced the necessity for uterine packing, with the risk of subsequent infection.

Sixty-four per cent of the obstetricians questioned advocated the use of some form

of posterior pituitary extract, usually obstetrical pituitrin in 1 c. c. doses, either alone or in combination with other oxytocics, at the end of the second stage of labor, by the intramuscular or subcutaneous route. We have found pituitrin and pitocin to be about equally effective for routine use. These preparations should not be used intravenously, for anaphylactic shock is a distinct possibility when this route is employed. Pitocin should be substituted for pituitrin in patients with hypertension, since pituitrin increases the blood pressure and decreases the urinary output.

In hospital practice an oxytocic may be given intramuscularly as soon as the anterior shoulder is delivered. Our experience has been that this routine shortens the third stage of labor, diminishes blood loss, and is particularly effective in that large group of cases in which the mechanism of the third stage of labor is imperfect because of the modern obstetric practice of sedation, anesthesia, and operative delivery. We also use ergonovine intravenously immediately after the delivery of the anterior shoulder whenever postpartal hemorrhage might be anticipated because of such conditions as prolonged labor, unusually deep anesthesia, a difficult operative delivery, or overdistention and subsequent atony of the uterus because of a large baby, twins, or hydramnios.

Enthusiasm for oxytocic drugs should not blind one to the dangers associated with their use at the end of the second stage, chiefly the possibility of placental incarceration as the result of hourglass or other contraction of the uterus. This danger can be averted by immediate expression of the separated placenta. Particular precautions should be taken in this regard when ergonovine is used intravenously, for under such circumstances its action is very rapid. Generally speaking, when the delivery is conducted in the home the use of an oxytocic should be deferred until after the placenta has been delivered.

Failure of the uterus to respond to one oxytocic is no contraindication to the use of another. Thus in postpartal hemorrhage due to uterine atony, ergonovine may be given intravenously and pituitary extract intramuscularly. Packing will be necessary only occasionally if bleeding is controlled by bimanual compression of the uterus until the effects of the oxytocic drugs are apparent.

THE THIRD STAGE OF LABOR IN THE COMPLICATIONS OF PREGNANCY

Certain abnormalities of pregnancy which may complicate the third stage of labor should be briefly discussed. They include placenta previa, abruptio placentae, placenta accreta, and uterine inversion.

Placenta previa: The management of the third stage of labor is of the utmost importance in patients with placenta previa, since the separation of the placenta from its abnormal site is often incomplete, and improper management may result in additional serious or even fatal blood loss. The best routine is to administer ergonovine (0.2 mg.) by vein as the anterior shoulder is delivered, and to effect delivery within the next 30-40 seconds. Rapid separation of the placenta usually follows at once, and gentle expression can safely be done. If separation and expression do not occur promptly, and if bleeding is at all profuse, manual removal of the placenta should be undertaken without further delay. If bleeding follows completion of the labor in any patient with placenta previa, the lower uterine segment should be investigated immediately, to determine whether lacerations have occurred from the trauma of labor or the operative procedures instituted to control hemorrhage.

Twenty-eight per cent of the 178 patients with placenta previa who were delivered by the vaginal route at Charity Hospital during the last decade required active measures for the control of hemorrhage in the third stage. More than one procedure was necessary in four cases. Manual removal of the placenta was done 14 times, repair of lacerations 20 times, and packing of the uterus and vagina 22 times.

Abruptio placentae: The third stage of labor and the first hours of the puerperium are likely to be the most eventful of the whole course in any case of abruptio pla-

centae. The best routine is to administer a potent oxytocic, preferably ergonovine by the intravenous route, as soon as the delivery of the fetus is completed. In the usual case the placenta is already separated and will promptly be expelled, along with numerous clots of blood. Prompt manual exploration of the uterus should be undertaken in any case in which expulsion of the placenta is unduly delayed.

Hemorrhage is usually controlled by the use of ergonovine in all cases in which the uterine musculature has not been too seriously impaired by hemorrhagic extravasation. If it continues, the uterus should be thoroughly packed with antiseptic gauze, after the placenta has been expressed. Since temporizing is not justified in so serious a complication, the next step, if hemorrhage continues unchecked, should be rapid hysterectomy, following an additional blood transfusion.

In the 186 patients with abruptio placentae who were delivered at Charity Hospital by the vaginal route during the last decade, the placenta was removed manually in nine cases and the uterus packed in 19 others because of continued bleeding after the third stage.

Placenta accreta: Placenta accreta, a complication of pregnancy in which the placenta is adherent to and inseparable from the uterine wall, should be suspected whenever bleeding does not occur in a case of retained placenta. The diagnosis is confirmed by failure to find a line of cleavage when manual removal is attempted. Immediate hysterectomy is the only possible treatment, since the decidua basalis layer is missing and the chorionic villi have invaded the uterine musculature.

The condition is rare. It occurred only three times in the 37,760 deliveries conducted at Charity Hospital in the last decade. One case terminated fatally.

Inversion of the uterus: Since uterine inversion is practically always the result of mismanagement of the third stage of labor, it stands in a different category from the abnormalities which have been discussed. It usually follows violent attempts at expression of the placenta by the Credé method, together with forceful traction on the cord, particularly if the uterus is greatly relaxed. It may also occur during manual removal of the placenta if the patient is under deep anesthesia and the uterus is markedly atonic. In a recent instance, observed at autopsy, a partial placenta accreta was apparently the etiologic factor.

As soon as the accident is discovered, reduction of the inversion should be attempted under anesthesia. If the attempt is successful, the uterus and vagina are tightly packed with sterile gauze, which should remain in place for 24 hours. If vaginal reduction fails, abdominal reduction or abdominal or vaginal hysterectomy should be resorted to without delay.

Three patients with acute inversion of the uterus were observed at Charity Hospital during the last ten years, two of whom died.

THE THIRD STAGE OF LABOR IN CESAREAN SECTION AND ABDOMINAL PREGNANCY

The management of the third stage of labor in patients delivered by cesarean section presents a different set of problems. Blood loss can be substantially diminished if the following procedure is carried out: After the head of the infant is delivered 1 c. c. of obstetrical pituitrin is injected directly into the uterine muscle and 0.2 mg. of ergotrate is given intravenously. The remainder of the delivery is slowly effected one or two minutes later. The uterus is thus given time to accommodate itself to the tremendous decrease in the size of the cavity which follows the extraction of the child, and the amount of hemorrhage and the duration of the operation are correspondingly decreased.

Puerperal morbidity in our experience has been substantially diminished by the use of another simple procedure, the placing of a tight sterile gauze intra-uterine pack, one end of which is carried through the cervix to be removed per vaginam after six to eight hours. Cervical drainage is thus established and postoperative morbidity is greatly decreased by the elimination of lochial retention. The uterine pack is also of value in controlling hemorrhage,

as we have observed in a series of cases of placenta previa and abruptio placentae.

The management of the placenta in abdominal pregnancy is not a simple matter under any circumstances. It is often impossible to remove it even partially because the placental tissue is densely adherent to the abdominal viscera. Unwise procedures may cause fatal hemorrhage. The more nearly full-term gestation is approached, the more frequently can placental detachment be achieved without serious hemorrhage, because a definite line of cleavage is present. In other cases the best plan is to remove the fetus, ligate the cord close to the placental surface, marsupialize the placental sac, and leave the placenta *in situ* to be absorbed.

In five cases of abdominal pregnancy, all of more than seven months' duration, observed at Charity Hospital during the last 10 years, the placenta was completely removed in two, left *in situ* in two, and partially removed in one. All patients recovered.

SUMMARY

1. The immediate and remote complications and abnormalities of the third stage of labor are responsible for a definite maternal mortality and for a high incidence of maternal morbidity.

2. Both morbidity and mortality are most often due to mismanagement of this very important stage of labor. Correct management is based on an accurate knowledge and comprehension of the normal mechanism of the third stage, in which recognition of placental detachment is essential.

3. A conservative, expectant policy is advocated for the management of the third stage of labor. It should not, however, be carried to the point of negligence by the postponement of active intervention when indications for it are present.

4. The management of certain pathologic conditions of the third stage is presented, together with the incidence and mortality of these complications at Charity Hospital of Louisiana at New Orleans during the decade ending January 1, 1940.

DISCUSSION

Dr. Edwin L. Zander (New Orleans): It is universally recognized that proper management of the third stage of labor is of utmost importance, yet discussion of this subject is infrequent and often passed over for more striking and unusual types of obstetric pathology.

Davis states that the third stage is divided into delivery of placenta, repair of lacerations and postpartal convalescence. I believe delivery of the placenta in the third stage is most important. With delivery of the fetus the fate and condition of the baby is decided ending the second stage. The fate of the mother, that is to say postpartum convalescence, is decided in the next thirty minutes after the end of the second stage. It has been my experience in seeing medical students, that they have a poor conception of the physiology of the third stage. I have noticed further that when senior students become interns there is an increase at first, in the amount of retained placentae and postpartum hemorrhage, resulting from undue tampering, pulling on the cord, and trauma. In clinical work we should stress prophylaxis of complications in the third stage. I have administered oxytocic at the end of the second stage. I believe this shortens the third stage. I do not believe that this routine has any deleterious effects. During the past 20 years I have had two retained placentae and could not blame this on the oxytocic used.

Another thing I would like to stress is the question of blood transfusions. They are more commonly used at the present time following delivery, particularly in women with much blood loss. The amount of loss of blood determines the condition of the patient in regard to infection which might follow delivery. In 1930 at Charity Hospital there was one blood transfusion to every 102 deliveries. In 1939 there was one to every 34 deliveries, showing, in my opinion, the importance of blood transfusions. These cases had comparative decreases in mortality and morbidity.

I believe that it is important that the cord should not be used for traction, and only as a guide for the delivery of placenta. No traction in delivery of the placenta is permissible. I believe the question of proper method of carrying out the Credé method for delivery of the placenta is very important in control of bleeding and other postpartal results. Contrary to some fellow obstetricians, I believe that in cases where the placenta is retained, probably a few hours, there is less harm in manual removal under aseptic conditions than leaving the placenta in.

Dr. Peter Salatich (New Orleans): A point in my experience which has helped a good deal in delivery of the placenta, is after the head is born, give the patient 1 c.c of pituitrin. It is surprising how easy the placenta is expressed. I have seen men deliver a baby and right away want to get the

placenta out; wait at least 10 to 15 minutes to allow the placenta to separate, providing the patient is not bleeding a lot.

Dr. E. L. King (New Orleans): I want to mention one or two points: One is manual removal of the placenta. I feel as Dr. Arnell, that a long wait is not wise. We have had cases where we waited 14 to 24 fours at the hospital, with fear and trembling I must admit; often the placenta would finally become detached spontaneously. During that time there might be bleeding and trickling from hour to hour, which is generally underestimated. I have adopted the policy that if the placenta is not delivered in a couple of hours, after using the Credé method, take it out manually.

In the matter of exploration of the uterus after operative delivery, that is something about which we should exercise some caution. I believe we should have definite indications for going into that uterus. Occasionally as Dr. Arnell has noted we find a ruptured uterus.

Dr. C. C. deGravelles (New Iberia): I think one of the most important factors in the third stage of labor and one of the things we should think most of is the conservation of the mother's blood. I believe too many of us, after the baby is born, wash our hands and sit back and just wait around until nature causes the clot to form and expel the placenta. I have found that it is a good idea after the baby is born, never to take my hands off the uterus. Frequently I have seen bad hemorrhages when that fact was disregarded. Especially is this true after a long tedious labor. Sometimes when the uterus was left to expel the placenta unaided I went back and found the uterus filled and considerable hemorrhage followed expulsion of the placenta. The best policy in my opinion is to keep the hands on the uterus all of the time until the placenta has been delivered.

There is one question I would like to ask in regard to the giving of pituitrin and that is whether it is used at the end of the second or the third stage?

Dr. R. E. Arnell (In conclusion): For the routine obstetric work in the home I believe that some form of oxytocic should be used, preferably administered at the end of the third stage. Administration at the end of the second stage requires perfect timing so that the placenta can be delivered at once after it has separated. If such perfect timing is not possible because the baby must be cared for, the cord tied, and so forth, uterine stimulants should not be given until placental delivery has been completed. Constant attention to the mechanism of the third stage will prevent most complications.

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MEDICO-MILITARY MATTERS

The examination of the draftees by the doctors who have so cheerfully and willingly volunteered their services is proceeding in the State of Louisiana smoothly and efficiently. In spite of the original request for one medical man for each draft board, it has been found, as it was thought at the time, necessary to increase materially the group of examining physicians so that very many more men have given their services than the list published last month in this Journal. As appointments are still being made and the present list is not complete, it will not be published until a later date. It was found impossible also to continue appointing men who were over the draft age, so a considerable number of the younger group of doctors have also been good enough to donate their services to this important cause.

Some of the officers in the Medical Reserve Corps have, to a limited extent, been called into active service. The Army has figured that it will need around 8,400 medical officers for the training period. There are now about 4,000 men on duty. Roughly these medical men are divided as follows: 1,200 from the regular Army; 1,600 from the National Guard and 1,200 from the Medical Reserve Corps. This will necessitate, when the program for the training becomes effective, approximately 4,400 more physicians. At the present time there are some 14,000 men in the Medical Reserve Corps and on the first of July there will be between 1200-1500 more available who have taken the R. O. T. C. training. About 13,000 of the physicians in the Medical Reserve Corps have the rank of Lieutenant or Captain. It will be these younger men for the most part who will be assigned to duty. The thousand or more men of higher rank may possibly be called only for special duty or for a short period of time.

In the meantime the Navy will need about 1,200 medical men for their increased program.

The figures given above are not official but they might be said to be semi-official. They were obtained from an officer assigned to the Surgeon General's office and are relatively authentic as of the date this was written. Of course they may be subject to change from day to day.

In the event of war, with full mobilization of the man power of this country, it is estimated that there will be needed 30,000 medical officers. This of course will disrupt civil practice to a very considerable extent. It is a problem which will have to be dealt with by Dr. Cole, Chairman of the Committee on

National Defense of the American Medical Association. In working out the difficulties that necessarily will arise, he will have to have the aid and assistance of the parish and district medical societies and their officers. Plans will have to be made to take care of the people at home should the present so-called emergency become an active one.

SYPHILIS IN LOUISIANA

Recently there has been sent out a report on the incidence of syphilis in the various states of this country, as well as in cities that have a population of over 200,000. The annual rate per thousand population of syphilis and gonorrhea in Louisiana is remarkably low. For the former disease it is 3.40 and for the latter 0.48. Contrasting neighboring states it is found that the rate for syphilis in Mississippi is slightly over 20 and for gonorrhea a bit over 14; in Texas it is 5.59 and 1.57 respectively. Arkansas has practically the same figures and Alabama is almost identically the same as Texas. On the other hand, in South Carolina the rate for lues is 10 plus, for gonorrhea 1 plus. The rate in the Southern states is, as would be expected for obvious reasons, higher than it is in the North, Northcentral and Western states. However, it must be borne in mind that the state health service in the Southern states for the most part is decidedly superior to that of the states elsewhere in the United States. The health service offered by Southern state boards of health is more complete and better organized than the majority of states in the country, consequently where there is good health service the reporting of syphilis and gonorrhea is carried out more carefully than elsewhere. Rather than being a reflection on Mississippi, the fact that they have the highest rate for these venereal diseases would further prove that it has one of the best health departments in the United States.

Of course, the actual rates are very much higher than the reported rates. Every physician knows that there is more gonorrhea than there is syphilis and yet the reported rates of gonorrhea are just one-third of syphilis, indicating very definitely that there is laxity in reporting gonorrhea and undoubtedly there is also laxity in reporting cases of syphilis. It is very doubtful that Mississippi has four times as much syphilis as Arkansas or Texas or even Alabama.

In New Orleans the rate is extremely low, 1.9 for syphilis per 1,000 population, 1.2 for gonorrhea. These figures are very much out of line with other cities in the United States, with the State as a whole and with venereal disease surveys of the incidence of syphilis in New Orleans. Clarke*, for example, found in New Orleans the rate of syphilis to be nearly three and a half times the reported rate and gonorrhea ten times more frequent than reported. The rate for syphilis in 1931 was estimated to be 12.45 per thousand. While the incidence of syphilis may have diminished considerably in the last nine years, it is doubtful if the rate could have dropped from 12 to slightly under 2. Definitely the New Orleans doctors are falling down on their job of reporting syphilis and gonorrhea, whereas the doctors throughout the State are very much more attentive to their duty in listing these two diseases.

PULMONARY EMBOLISM

Surgeons and obstetricians are well acquainted with the dangers of pulmonary embolism and of the complications that occur as result of embolism of the pulmonary artery after operation. On account of the increasing number of roentgenograms of the chest being made daily, the roentgenologists also have shown marked interest in the study of pulmonary infarcts. On the other hand, writes White,* the medical man has not been concerned to any great extent with the medical aspects of the problem. He points out, in a recent paper, that he himself for many years did not appreciate the frequency of pulmonary embolism and

*Clarke, Walter: The burden of syphilis and gonorrhea in New Orleans, New Orleans M. & S. J., 84:163, 1931.

*White, Paul D.: Pulmonary embolism and heart disease, Am. J. Med. Sci., 200:577, 1940.

infarction and it has been only within the last comparatively few years when he has made particular efforts to discover this relatively common complication of heart disease that he has realized it is a fairly frequent condition.

In the cases that are reported by this observer, the great majority, as would be expected in the practice of a cardiologist, were dependent upon a preceding heart lesion. In the 370 cases of pulmonary embolism and infarction studied after death at the Massachusetts General Hospital, 40 per cent were postoperative with 59 per cent of infarction; 30 per cent were cardiac with 90 per cent infarctions and 30 per cent were non-cardiac medical cases with 72 per cent of infarcts. In other words, infarction is very much more likely to occur in the heart patients but the percentage relation between the cardiac and non-cardiac patients is about equal.

Some of these cases without cardiac disease resemble very closely cases of heart disease. These were the patients who had acute cor pulmonale, whose right ventricle was dilated, the neck veins were engorged and other symptoms of right heart failure were present.

It is to be expected that in the instance of complicated heart disease associated with congestive heart failure with the usual signs at the base of the lungs, the physical expressions of pulmonary infarction may be completely obscured. The physician naturally expects or diagnoses bronchopneumonia when the patient develops fever and leukocytosis, plus shortness of breath, relative marked prostration and particularly a tachycardia which may be associated with chest pain, cough and hemoptysis. The diagnosis obviously is difficult but there is this to be remembered that in a cardiac patient with congestive failure who does not respond to treatment this possibility of pulmonary infarction should be in the mind of the practitioner. The group of symptoms may simulate coronary thrombosis.

In so far as treatment is concerned, oxygen plus morphine, plus atropine, plus digitalis, are the best drugs to administer. The patient should be kept absolutely at rest and this rest should be enforced not only upon the patient but also upon the doctor. Sometimes patients are killed by too great attentiveness to treatment by the physician trying to do too much. Fortunately the acute symptoms subside promptly and the anticipated misdiagnosed pneumonia, with its course of a week or two does not materialize because the man is either dead or on the road to recovery within forty-eight to seventy-two hours.

HOSPITAL STAFF TRANSACTIONS AND CLINICAL MEETINGS

SOUTHERN BAPTIST HOSPITAL
NEW ORLEANS

The regular monthly meeting of the Clinical Staff was held in the staff room of the hospital at 8 p. m. on November 26. The program consisted of the following presentations: "Premature Separation of placenta with Case Report," by Dr. E. A. Ficklen; "Solitary Kidney with Marked Hydronephrosis, with Case Report," by Drs. M. M. Green and H. H. Russell; "Three Interesting, Odd Cases" by Dr. W. B. Clarke; "Death Report", by Dr. Bert DeBord.

C. L. Cox, M. D., Chm.

CHARITY HOSPITAL
MEDICAL DIVISION
NEW ORLEANS

The regular monthly meeting of the Medical Section of the Charity Hospital Visiting Staff was held on Tuesday, October 15, 1940, at 8:00 p. m. in the auditorium of the hospital. The meeting was called to order by the Chairman, Dr. T. A. Watters.

The program consisted of the following, presented by members of the staff of the Tulane Division: A pathologic presentation of "Bronchiogenic Carcinoma" by Dr. H. J. Schattenberg; "A Case with Brachial Plexus Lesion" by Dr. R. H. Turner; "A Case Presentation" by Dr. W. A. Sodeman; and "A Case of Spontaneous Hypoglycemia" by Dr. R. P. Vieth.

Bronchiogenic Carcinoma (Dr. H. J. Schattenberg): The patient was a white male, aged 52, who was admitted to the hospital on August 17, 1940, with the chief complaints of anorexia, loss of weight and weakness. He was in apparent good health until two weeks prior to his admission, at which time he developed a cough which was productive of mucoid sputum. There was no history of

chest pain or hemoptysis. He claimed he had lost 35 pounds in weight from the beginning of the present illness until admission to the hospital. He has been mildly dyspneic. Temperature, pulse, and blood pressure were normal on admission. The respiratory rate was 25 per minute. There were no palpable lymph nodes in the neck. The thorax was symmetrical. A few moist rales were heard at the right base posteriorly. The blood picture values were 65 per cent hemoglobin and 3,400,000 red blood cells. The sputum examination was negative. Bronchoscopy was performed and the right and left bronchi were found to be filled with thick, creamy pus. No tumors were noted. X-ray of the chest was interpreted as showing metastatic tumor nodules in both lungs. During the patient's stay in the hospital he continued to cough, became progressively weaker and more hoarse. He died forty-two days after admission.

The gross and microscopic pathologic diagnosis at autopsy was primary carcinoma of the lung with metastasis throughout both lungs.

In the discussion the various hypotheses advanced in an attempt to explain the mode of metastasis of a primary tumor to other parts of the same lung or to the opposite lung were presented. Several slides were shown illustrating gross, microscopic and x-ray findings in this and similar reported cases.

A Case with Brachial Plexus Lesion (Dr. R. H. Turner): The patient, whose case I wanted to present, has left the hospital. It is a case of a white male, aged 39, a farmer, who has had five admissions to the hospital in the last year and on each admission, except the first, something was brought out which aided in the development of a highly diagnostic clinical picture. The first admission was for skin lesions on the face. In February 1940 he had what he thought was influenza with generalized weakness and fever. He became quite sick and stayed in bed for a week. Some doctor said he had pneumonia and another sneered at this diagnosis and thought it was influenza. He was never well after this illness. In two weeks he developed pain in the right shoulder which extended to the right arm and hand. During the next few months the pain became extremely severe and radiated down the arm to the ulnar side to the ring finger more than any other portion of the hand. He was admitted in April to a surgical service, still complaining of this pain. He was diagnosed at that time as having peripheral neuralgia. The intern noted that the left pupil was larger than the right. On examining the lungs Dr. Boaz states that he suspected pathology in the apices of the lungs, particularly the right. The patient stayed in the hospital only a few days, then came back in May on the orthopedic service. At that time the right arm was weaker and smaller than the left. There was no note made of pupils or difference in sweating. He still complained chiefly of pain in the shoulder and chest in the region of the fifth rib. He was readmitted in August, at which time I saw him for the first time. The right pupil was smaller, there was drooping of the right lid but no difference in sweating noted in the two portions of the face. His right shoulder drooped and he carried his right arm in twisted position and his head was twisted to the right. The entire right hand and arm were extremely weak, sensitive to touch but did not sweat. There was obvious atrophy of the muscles of the arm and hand. (The suggestion was made at that time that he had a tumor in the apex of the right lung which involved the brachial plexus.) There was a mass behind the clavicle and the sternomastoid but the muscle was so tense that it was difficult to be certain of its nature. It was noticed that the x-rays made in April showed little, but the diagnosis was made of old tuberculosis at both apices. X-rays made in June showed a definite clouding in the right apex but there was no abnormality of the bones. X-rays made in August showed a greater apical density but no lesion of bone, but during the month of September, during an additional admission, there was demonstrated quite clearly destruction of a portion of the second rib on the right. There was one part that showed some erosion of the lateral surface of two vertebrae.

This patient exhibits all the cardinal features of Pancoast's syndrome. Since Pancoast's report in 1932, there have been a great number of reports in the literature, many of which I have read, and I am rather impressed by the stereotyped findings. Apparently Pancoast made one mistake as there were no autopsy findings in his series and only three biopsies, and in spite of this fact he stated that these tumors did not arise from the lung. It is clear that the nature of the tumors is multiple. Most of them are primary carcinomas of the lung; others do arise from the brachial cleft structures, or from the sympathetic nervous system. There are no accurate figures on the total incidence but it must be quite rare. In a Philadelphia series of 6,000 autopsies there were 500 malignancies, 32 were primary carcinoma of the lung. There were no instances of this syndrome. It would seem to cause death less frequently than once in 6,000 deaths. It seems to occur less frequently than once for 30 carcinomas of the lung, which makes it a fairly rare syndrome. It occurs about equally on the two sides. One of the characteristic features is that patients do not commonly have lung symptoms, such as cough, hemoptysis or signs of infection in the lung, as do other bronchiogenic carcinomas so frequently. The ones that occurred on the left have been more commonly associated with pleural effusion and erosion of the ribs farther down around the apex of the lung. It may be purely accidental that the difference between the right and left is striking. Of course, a great deal has been written about why they happen to occur so far in the periphery of the lung and why they invade the neck. I have seen no good explanation. If there were lymphatic channels in apical pleural adhesions, this might be a partial explanation. The

condition is nearly always associated with extreme suffering. The pain is intense, long and unremitting and not relieved by anything that is available. The use of radiotherapy has been completely unsuccessful. No one who has tried it recommends its use. So far, I would say that the syndrome is of great interest because of the sharply drawn clinical picture, because of the rather puzzling features of the pathogenesis, and it is terrifying because of the suffering it involves and the hopelessness of the therapy.

DISCUSSION

Dr. Bayley: I have seen this same syndrome caused by actinomycosis in a patient who was admitted to the hospital and died in about two months' time. For a while we considered it as due to a tumor but a sinus developed and actinomyces were found.

A Case Presentation (Dr. W. A. Sodeman): A negress, age 29, was in good health until three weeks before admission when she developed a head cold followed by non-productive cough. In several days she became short of breath and developed a sharp pain in her left chest posteriorly. She thought she had had fever for about a week before admission. Past and family histories appeared unimportant. Physical examination revealed a febrile patient who appeared acutely ill. She was dyspneic with a hot, dry skin, and had evidence of rhinitis and carious teeth. The findings of interest were in the chest. There was some dulness in the left base posteriorly with absence of breath sounds in the same area. The cardiac rhythm was regular, the heart was enlarged to the left, pulse was 106 per minute and parodoxical in type. Blood pressure was 110/78. X-rays of the chest on the way to the ward showed a tremendously enlarged cardiac shadow. Venous pressure varied from 22 to 25 cm. of water. The abdomen showed slight left upper quadrant tenderness and the pelvis was not remarkable.

Pericardial tap produced 260 c. c. of blood-tinged fluid with a specific gravity over 1.020, and no organisms on smear or culture. Sections made of the centrifuged sediment showed red blood cells and occasional lymphocytes. Tenderness developed over the left lung base and again 440 c. c. were withdrawn from the pericardium by anterior tap. Increase in the dulness posteriorly and extension into the axilla led to the diagnosis of pleural effusion and 600 c. c. of fluid, identical in character with the pericardial fluid obtained anteriorly, were withdrawn.

The Mantoux test was positive in dilution of 1:10,000 but not in dilution of 1:100,000. The white blood cells of the blood never exceeded 8,100 per cubic millimeter. The blood settled 18 mm. in 35 minutes. The urine showed occasional casts and a slight amount of albumin. Sputum was negative. The Wassermann was repeatedly positive. Blood cultures were negative, as were agglutinations for tularemia, undulant fever, typhoid, and paratyphoid. Blood glucose was 78 and urea 14. Fluoroscopy of the heart showed a greatly enlarged shadow with a contour that fitted well with the diagnosis of pericardial effusion and with feeble pulsations. The esophagus was not displaced. The electrocardiogram showed no ST shift, but the T-waves were inverted in all four leads.

She had had fever almost continuously since admission. It varied from 103° to normal and was not affected by 120 grains of sodium salicylate daily for a week. At present she weighs 105 pounds.

The diagnosis of pericardial effusion is obvious. The cardiac shadow obscures the lower left lung field in the x-ray and the presence of pleural fluid is doubtful. It is possible that the fluid obtained by posterior tap was pericardial. Injection of air at the time would have settled this point but unfortunately was not done.

Since the fluid is obviously an exudate, we must consider the causes of pericarditis with effusion. The chief causes are: (1) pneumonia, influenza, and other infections in proximity to the pericardium, (2) tuberculosis, (3) rheumatic fever, (4) disseminated lupus, (5) trauma. The character of the fluid rules out the purulent types and especially those due to organisms easily recognized by fluid culture and examination. Therefore, tuberculosis, rheumatic fever, and lupus are the most likely possibilities. There is little to favor the last in the absence of renal involvement, arthritis, and skin manifestations. All patients, however, do not have skin manifestations.

Tuberculous pericarditis is most frequent in the negro and 90 per cent occur in males. As in this patient, past and family histories are usually negative. The mode of onset is, however, usually insidious. Evidences of cardiac tamponade develop. Significant murmurs are absent. About 30 per cent develop Ewart's sign. It is most frequent in older age groups, only 25 per cent occurring from 20 to 30 years. Dyspnea and cough are common, but sputum and hemoptysis rare. Again, as in this patient, the white blood count is in normal range, and the fluid is often serosanguineous.

Rheumatic pericarditis usually develops in children and frequently a past history of rheumatic arthritis, other evidences of the rheumatic syndrome, or rheumatic heart disease can be obtained. Leukocytosis is expected and in the effusion polymorphonuclears predominate. With pancarditis the heart is usually dilated and replacement of fluid with air demonstrates an enlarged heart. In tuberculous pericarditis the same procedure usually shows a normal sized heart. Without a past history of rheumatic fever and without valvulitis, rheumatic pericarditis may be otherwise difficult to diagnose. Salicylates are said generally not to affect the heart. They affect especially the exudative manifestations of rheumatic fever and reports have

shown reductions in pericardial effusion and temperature, reduction in venous pressure, and relief of symptoms. Such reports are found especially in the French literature. These responses were not found in the present patient.

You can see from what I have said that I think the diagnosis is tuberculous pericarditis. The diagnosis of disseminated lupus is a remote possibility. There may be some other diagnosis I have not mentioned, but I cannot think what it might be. If any have arisen in your minds, I would like to hear them.

DISCUSSION

Dr. Bayley: I would like to say the symptoms which Dr. Sodeman discussed are more or less characteristic of tuberculous pericarditis with a rapid accumulation of the large quantity of fluid. This mass of fluid is not usually seen in rheumatic pericarditis. I was just wondering if replacement with air would not keep this cavity dry.

Dr. Sodeman: I think that air should be injected into the pericardial cavity and that it may be of some importance in preventing an adherent pericardium. What Dr. Bayley says about the size of effusion in rheumatic fever and tuberculosis is true. Of course, in tuberculous effusion, where the effusion is very great, tap has to be done to relieve symptoms. I only wish that some air had been put in at the tap posteriorly and we would have known if she had fluid in two cavities instead of one.

A Case of Spontaneous Hypoglycemia (Dr. Richard P. Vieth): This patient is a three year old colored female, who was first seen in the Outpatient Department of Charity Hospital on June 4, 1940. The history is that early in March of this year, the patient suddenly had a "falling-out" spell, lost consciousness, and could be aroused only with difficulty. She remained in this semiconscious condition for about half an hour. After that time, she had repeated attacks similar to the one I have just described until the onset of what the mother called convulsions on June 4. On questioning, the mother revealed the fact that the convulsions consisted of stupor, with twitching of the hands and arms, and peculiar movements of the jaws, resembling swallowing motions.

The past history of the patient is not significant except in so far as the diet is concerned. She was breast fed until she was one year old and then she was given Magnolia milk and vegetables. She also has had cereals and vegetables in her diet, but very little in the way of animal fats and proteins.

The family history is also negative. The child was seen several times in the clinic, but there was no change in her condition. She continued to have repeated attacks, such as I have described, occurring several times a day. On September 20, she was hospitalized for more thorough study. Physical examination was entirely negative. The laboratory data, with the exception of the blood chemistry, were also negative. On five different occasions, the fasting blood sugar values were 133, 58, 60, and 48 mg. per cent. A glucose tolerance test revealed the following values: fasting blood sugar, 58 mg. per cent; one-half hour, 62 mg. per cent; one hour, 62 mg. per cent; two hours, 62 mg. per cent; three hours, 54 mg. per cent. A spinal puncture was done and was negative except for the fact that the spinal fluid sugar content was too low to be read.

During the patient's stay in the hospital, she has had many attacks. They are usually precipitated by the withholding of food for only a few hours beyond her usual mealtime. In every instance, she is restored immediately and dramatically to complete consciousness and well being by the administration of orange juice.

From the clinical picture and laboratory findings, we feel justified in making a diagnosis of spontaneous hypoglycemia in this case. As yet, the cause has not been determined.

If extra-pancreatic causes for the hypoglycemia can be ruled out, and if she does not respond after a fair trial to the usual dietary and medical measures, we believe that a laparotomy is indicated.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

December 2. Orleans Parish Medical Society Board of Directors, 8 p. m.
Clinico-pathologic Conference, Hotel Dieu, 8:15 p. m.

December 3. Eye, Ear, Nose and Throat Staff, 8 p. m.
Chaillé Memorial Oration, Hutchinson Memorial Auditorium, 8 p. m.

December 4. Clinico-pathologic Conference, Charity Hospital Morgue Amphitheater, 1:30 p. m.
Hutchinson Memorial Clinic Staff, 8 p. m.
Mercy Hospital Staff, 8 p. m.

December 5. Clinico-pathologic Conference, Touro Infirmary, 11:15 a. m. to 12:15 p. m.

December 9. Orleans Parish Medical Society, 8 p. m.

December 10. Eye, Ear, Nose and Throat Staff, 8 p. m.

December 11. Clinico-pathologic Conference, Charity Hospital Morgue Amphitheater, 1:30 p. m.
Touro Infirmary Staff, 8 p. m.

December 16. Hotel Dieu Staff, 8 p. m.

December 17. Charity Hospital Medical Staff, 8 p. m.

December 18. Clinic-pathologic Conference, Charity Hospital Morgue Amphitheater, 1:30 p. m.
Charity Hospital Surgical Staff, 8 p .m.
The Orleans Tuberculosis Hospital Staff, 8 p. m.

December 19. Clinico-pathologic Conference, Touro Infirmary, 11:15 a. m. to 12:15 p. m.

December 20. I. C. R. R. Hospital Staff, 12 noon.
New Orleans Dispensary for Women and Children Staff, 8 p. m.

December 24. Baptist Hospital Staff, 8 p. m.

December 26. Clinico-pathologic Conference, Touro Infirmary, 11:15 a. m. to 12:15 p. m.

December 27. L. S. U. Faculty Club, 8 p. m.

During the month of November, the Society held one regular scientific meeting and a special meeting with the U. S. Marine Hospital Staff. The following program was presented at the regular meeting:

1. Biographical Sketch: Reflections on Dr. Edmond Souchon By Dr. Isidore Cohn
2. Case Report: Intramedullary Aneurysm of the Cord
   By Drs. D. H. Echols and R. G. Holcombe, Jr.
3. Symposium on Recent Advances in Vitamins
   a. Chemistry of Vitamins
      By Invitation Dr. Arthur O. Kastler
   b. Medical Aspects By Dr. Samuel B. Nadler
   c. Surgical Aspects By Dr. Michael DeBakey

At the special meeting the following program was presented:

1. Sulfonamides in the Treatment of Gonorrhea in the Male
   By Dr. John A. Trautman, Past Assistant Surgeon
2. Case Report: Typhoid Carrier
   By Dr. William L. Smith, Senior Surgeon.
3. New Technic for Repair of Inguinal Hernia
   By Dr. John D. Lane, Jr.

Nomination of officers of the Orleans Parish Medical Society and delegates to the Louisiana State Medical Society, for 1941, were read by the Secretary at the regular meeting.

The following physicians were elected to active membership in the Society: Drs. Rafael F. Ayon, Joseph M. Brocato, J. Fred Dunn, Elmer E. W. Kramer, Evelyn B. Nix, Ruth L. M. Shushan, Carl J. W. Wilen, Hippolyte P. Marks and Edward A. Jurgelwicz; to intern membership, Julius W. Davenport, Jr., Walter P. Diaz, George G. Gill, Courtland P. Smith, Sidney Stillman, and Samuel Zurik.

NEWS ITEMS

Dr. Henry B. Alsobrook spoke before the Tangipahoa Parish Medical Society, Hammond, October 3. The title of his paper was "Office Gynecology."

Dr. O. W. Bethea discussed immaterial cardiac conditions at a clinical staff meeting of the Baptist Hospital, October 22. Drs. Robert Bernhard and George E. Barnes presented a study of a case of rheumatic fever. Dr. W. R. Hardy discussed a case of cholelithiasis complicated by peptic ulcer of the duodenum. Dr. C. L. Cox presided at the meeting.

Drs. Michael DeBakey and Alton Ochsner presented a paper on "Achalasia of the Esophagus" at the American College of Surgeons, Chicago, October 24.

Drs. C. Gordon Johnson, Isaac W. Kaplan, Samuel Karlin, John G. Menville, and Lawrence H. Strug were initiated as follows into the American College of Surgeons at a convention in Chicago, October 21.

Dr. Leon J. Menville has been appointed part time head of the radiology department at Charity Hospital. Dr. W. D. Phillips was appointed chairman of the medical committee.

Dr. Alton Ochsner gave talks before the Montgomery County Medical Society at Montgomery, October 15, the North Mississippi Medical Society at Oxford, October 16, and the Calumet Branch of the Chicago Medical Society on October 24.

Dr. Walter J. Otis was elected a councilor of the Southern Psychiatric Association at a meeting of this organization at Jacksonville, October 21-22. Drs. Gilbert C. Anderson and Charles S. Holbrook also attended this meeting. Dr. Anderson presented a paper on "Relationship between Some Neuropsychiatric Conditions and Oral and Dental Lesions." Dr. Holbrook presented a paper on "Psychosis in Identical Twins."

Dr. W. H. Perkins was toastmaster at a luncheon recently given by the Tuberculosis and Public Health Association of Louisiana in honor of executives of the National Tuberculosis Association.

Dr. Howard Mahorner addressed the Hartford Medical Society at Hartford, October 7. The title of his paper was "Surgery of the Venous System." Dr. Mahorner also presented a paper entitled, "Recent Advances in the Surgical Management of Goiter," before the Gulf Coast Clinical Society at Gulfport, October 17-18.

Dr. John T. O'Ferrall attended the meeting of the Clinical Orthopedic Society at Milwaukee, October 18, and Madison, October 19.

THE GOLF TOURNAMENT

Dr. D. J. Farley is the new golf champion of the Orleans Parish Medical Society, succeeding Dr. L. R. DeBuys. Dr. Farley won the group's annual

tournament Wednesday at the Metairie Golf Club with low gross 81.

Dr. W. C. Rivenbark, chairman of the golf committee, announced the tournament winners as follows:

Low Gross—Dr. D. J. Farley, 81; Dr. Val Fuchs, 84; Dr. H. Meyer, 85.

Low Net—Dr. Allan Eustis, 71; Dr. J. S. Webb, 72; Dr. H. A. Thomas, 73.

Blind Holes—Dr. F. Cato, 33; Dr. K. Brau, 37; Dr. L. R. DeBuys, 37.

Poker Hand on First Nine—Dr. E. L. King, Dr. F. Boyce.

Poker Hand on Second Nine—Dr. J. H. Musser, Dr. E. L. Zander.

Nearest No. 12 Hole—Dr. Jack Strange, 30 feet 4 inches.

Nearest No. 17 Hole—Dr. W. C. Rivenbark, 4 feet.

High Score—Dr. M. P. Schwarzenbach, 115.

Kickers—First, Dr. C. H. Weinberg, 81; second, Dr. W. R. Hardy, 79; Dr. J. K. Stone, 79; Dr. R. F. Sharp, 79; Dr. L. A. Fortier, 79; third, Dr. E. H. Lawson, 84; Dr. Ed Faust, 84; Dr. R. E. de la Houssaye, 84; Dr. C. B. Odom, 84.

TREASURER'S REPORT

Bank balance September 30, 1940	$ 3,910.57
October credits	370.47
Total credits	$ 4,281.04
October expenditures	615.08
Actual book balance October 31, 1940	$ 3,665.96

Edwin L. Zander, M. D., Sec.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	

OUACHITA PARISH MEDICAL SOCIETY

The regular monthly meeting of the Ouachita Parish Medical Society was called to order November 7, at 7:30 p. m., by Dr. D. T. Milam, president. The minutes of the meeting of October 6 were read and approved.

The president announced that due to the number of business matters to be discussed there would not be any scientific program.

The Committee on the Mutual Hospital Association gave its report and a letter from Mr. Colvin was read. Mr. Colvin asked permission from the society to ask each doctor the number of calls and operations and the amounts collected on employees and their families of the Brown Paper Mill. It was so moved by Dr. Simonton, seconded by Dr. Graves and carried.

Letters from the New Orleans Graduate Assembly, the L. S. U. Extension Service and the National Physicians' Committee were read.

Dr. George Snellings presented the report of the Committee for the Banquet honoring Dr. Faulk. The committee has invited Dr. J. T. Nix to be the speaker and he has accepted. Invitations will be extended to representatives of the adjoining parish medical societies.

The application of Dr. J. H. Parrott was read and referred to the Membership Committee.

Dr. Milam announced that this would be the last meeting at which he would preside, because he had been called for active duty. Dr. Hamilton moved that the society give Dr. Milam a rising vote of thanks for the efficient and excellent president he has made. This was done.

L. L. Titche, M. D., Sec.

MOREHOUSE PARISH MEDICAL SOCIETY

The organization convened on Tuesday, November 19, to pay honor to the councilor of their district, Dr. W. A. Rodgers. There was a large attendance of doctors, not only from Morehouse Parish but many from Monroe. A delicious barbecue dinner was served at the Country Club, and was followed by talks from several of the local members as well as from Dr. P. T. Talbot, secretary-treasurer of the State Society, Dr. D. N. Silverman, councilor of the second district, and Dr. J. H. Musser, president of the State Board of Health.

Dr. R. B. Robins, of Camden, councilor of the fifth district of the State of Arkansas, was a guest of the society, and presented a paper on "Head Injuries," illustrated with lantern slides.

THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

The fifth annual meeting of the New Orleans Graduate Medical Assembly will be held March

3-6, at the Roosevelt Hotel, followed by clinics in the hospitals of New Orleans on March 7. Eighteen guest speakers, each one an outstanding leader in his special field, will contribute liberally to the program and will be available for informal discussions with those who attend.

It is especially significant that the Assembly has enjoyed an increase in attendance each year, last year's registration being over one thousand. Many of the registrants were physicians who attended previous meetings which is very gratifying to the sponsors because it indicates that the previous programs have supplied just what the busy doctor needs in the way of an intensive refresher course.

The success of the previous assemblies, together with the experience gained in planning courses of this type, has been the inspiration to introduce additional features each year. The regular four-day program will be followed by clinics in the local hospitals on March 7, conducted by members of the Assembly in medicine, surgery and allied specialties. These clinics will add greatly to the interest and value of the meeting because of the wealth of material available in New Orleans.

In addition to the featuring addresses and round-table discussions by eighteen of the country's foremost medical educators, the scientific and technical exhibits which are being sponsored by the two medical schools, individual members and the leading ethical manufacturers of drugs and equipment, will be more elaborate than ever.

Following the program in New Orleans will be the second annual Post-clinical Cruise to Havana, Panama, Canal Zone, and Honduras. The cruise is scheduled for March 8 and a most interesting, instructive and relaxing itinerary is planned for the sixteen days. Talks will be given aboard, followed by general discussions and medical motion pictures. Visits will also be made to the outstanding hospitals in Havana and the Panama Canal Zone, as well as the United Fruit Company hospitals in Honduras.

The Assembly offers five full days of medical education, combined with recreation in the interesting city of New Orleans. The registration fee of $10.00 covers all features, including four daily round-table luncheons as well as the smoker. The cruise is, of course, additional and if descriptive literature and booking information is desired write to the secretary of the New Orleans Graduate Medical Assembly, Room 105, 1430 Tulane Avenue, New Orleans.

AMERICAN PUBLIC HEALTH ASSOCIATION

The recent meeting held October 7 in Detroit, the sixty-ninth annual meeting, was attended by 3,187 physicians, sanitarians, statisticians and others connected with public health work. Dr. W. S. Leathers, of Nashville, Tennessee, was elected president of the organization, whose next meeting will be held in Atlantic City in October, 1941.

ARMY MEDICAL LIBRARY

The librarian of the Surgeon General's Library requests that reprints of articles published in current medical literature be sent to the Army Medical Library.

A. M. A. BROADCASTING

Attention is again called to the series of broadcasts presented by the A. M. A. over the national broadcasting chain. The broadcasts will take place every Wednesday until June 3, 1941, at 9:30 p. m., over the Blue network.

MEDICAL AND SURGICAL SUPPLY COMMITTEE

This organization actively is soliciting aid for the medical men of Great Britain. Instruments, surgical supplies and all types of equipment are needed. The medical men in Louisiana who have some of these materials which they no longer need are urged to send them to one of the members of the regional committee of the state. These men are Drs. Rudolph Matas, D. N. Silverman, Alton Ochsner, and J. H. Musser, of New Orleans; and A. A. Herold, of Shreveport.

SOUTHERN MEDICAL ASSOCIATION

At the recent meeting of the Southern Medical Association several New Orleans physicians were chosen as sectional officers. Dr. T. A. Watters was named vice-chairman of the section on neurology and psychiatry; Dr. Maurice Lescale, vice-chairman of the section on proctology; Dr. Merrill Beck, secretary of section on anesthesia; Dr. John T. Sanders, secretary of section on gynecology, and Dr. Joseph W. Reddoch, chairman of the section on obstetrics.

There was a large attendance among the doctors from New Orleans at this very successful meeting.

Dr. L. A. LeDoux, councilor from Louisiana, was appointed to the Executive Committee of the council for a three year term.

The third annual meeting of the American Academy of Dermatology and Syphilology will be held at the Palmer House in Chicago, December 8-11.

NEW ITEMS

Dr. Charles James Bloom taught at the Southern Pediatric Seminar in Saluda, North Carolina during the past summer.

Dr. Ambrose H. Storck recently participated in a panel discussion on abdominal traumas which was held in conjunction with the Clinical Congress of the American College of Surgeons in Chicago.

Assistant Surgeon G. E. Parkhurst has been transferred from the U. S. Marine Hospital to the Quarantine Station at New Orleans.

Assistant Surgeon Alfred L. Holloman has been ordered to the Coast Guard Training Station at New Orleans.

INFECTIOUS DISEASES IN LOUISIANA

The morbidity report of the Bureau of Epidemiology of the Louisiana State Board of Health for the week ending October 26, the forty-third week of the year, showed the following diseases reported in numbers greater than ten: 144 cases of syphilis, 35 of cancer, 34 of pulmonary tuberculosis, 25 of pneumonia, 21 of malaria, 20 each of diphtheria and gonorrhea, 18 of undulant fever, and 13 of chickenpox. During this week there were reported three cases of poliomyelitis from Orleans Parish. The remarkably large proportion of patients with undulant fever came practically in toto from Caddo Parish. The following week syphilis again led all reported diseases with 256 instances, followed by 30 of cancer, 29 of pulmonary tuberculosis, 24 of gonorrhea, 21 of diphtheria, and 12 each of malaria, pneumonia, and whooping cough. For this week there were reported seven cases of poliomyelitis throughout the state, three arising in Caddo. For the week which terminated November 9, the following commonly reported contagious and infectious diseases appeared: 145 of syphilis, 30 of cancer, gonorrhea, and uncinariasis, 29 of pulmonary tuberculosis, 23 of pneumonia, 13 of trachoma, and 12 of diphtheria. In this week three additional cases of poliomyelitis were reported from Caddo, three from Orleans and one from Calcasieu. For the last week for which the reports are available it is to be noted that there were tabulated 180 cases of syphilis, 37 of pulmonary tuberculosis, 32 of cancer, 23 of gonorrhea, 20 of pneumonia, 10 each of trachoma and diphtheria. In this week there was an additional case of poliomyelitis reported in Caddo and two in Orleans.

HEALTH OF NEW ORLEANS

The Bureau of the Census, U. S. Department of Commerce, reports that for the week closing October 19, 1940, there were 136 deaths in the city of New Orleans, of which 79 were white and 57 negro and 13 occurred in children under one year of age. This report was comparable to the report of the previous week. For the next week, ending October 26, there were 145 deaths, divided 82 white and 63 negro and 14 infants. There was a drop of 22 deaths for the week ending November 2. Of the 123 people who died in New Orleans this week, 71 belonged to the white population and 52 to the negro. The infant death rate was unusually high, there being 16 infant demises. A sharp increase 160 deaths being recorded, shared on the percentage basis about equally between the two races. There were 96 white deaths, 64 negro, and again a high infantile incidence of death, 19 in number.

JOSEPH A. ESTOPINAL
(1876-1940)

The death of Dr. Joseph A. Estopinal on October 9, 1940, removed from the ranks of organized medicine a member of long standing.

Dr. Estopinal was born in 1876 and graduated from Tulane University in 1899.

HENRY EDWARD MENAGE
(1872-1940)

The many friends of Dr. Henry E. Menage were shocked to hear of his death on October 23, 1940. Dr. Menage was one of the first specialists in dermatology in the city and for many years has had the reputation, local and throughout the United States, as a man of distinction in his branch of medicine.

A charming person, Dr. Menage will be mourned by his many friends and patients.

DR. A. L. LEVIN

The following memoir was passed at a recent meeting of the Staff of Hotel Dieu:

The Maker has called from our midst a friend, a fellow worker, an enthusiast in his chosen profession and specialty. He passed on in the height of life and as one who was on the brink of the promised land of fame and security.

"He is not dead, who lives in the hearts of his fellowmen."

Be it resolved that we make our records complete by inscribing upon them the name of a doctor, a scholar, a philosopher and a man, who, though tested in the fire of adversity in youth, extreme sorrow in middle age, carried on to eminent success in spite of the fact that he was not strong in body.

His indomitable will carried him through.

By resolving, we but make known our affection for a fellow worker. We want the world to know how near and dear he was to all of us. His many years of association with the Sisters of Charity, his kind acts to Sisters of all Orders. His willingness to help whether it be for the advancement of medicine or social work in the institution. We hope in a small measure to bring these facts to light. His honors are the result of an unceasing work and application and should be an inspiration to the youth in medicine.

His earnestness and original works are shown by his many articles and papers written and instruments devised. His skillful handling of those given to him by other hands make him an outstanding specialist among doctors and specialists.

Our city, our hospitals will know a void in the passing of this specialist, who while deeply religious, knew no creed, race or color in the administration of his work and his daily life.

Be it further resolved that a copy of these resolutions be sent to the family of Dr. A. L. Levin

as a symbol of our appreciation of sorrow at his death and as an expression of profound sympathy.

Further that a copy be sent to the Orleans Parish Medical Society, Louisiana State Medical Society, The Southern Medical Association, National Gastroenterological Association, American Society of Tropical Medicine, American Medical Association, and the Louisiana State University Medical Center.

(Signed): Louis Levy, M. D.
Chairman.

WOMAN'S AUXILIARY

Louisiana State Medical Society

President—Mrs. Roy Carl Young, Covington.

President-elect—Mrs. Aynaud Hébert, New Orleans.

First Vice-president—Mrs. Joseph E. Heard, Shreveport.

Second Vice-president—Mrs. W. A. Ellender, Houma.

Third Vice-president—Mrs. M. H. Foster, Alexandria.

Fourth Vice-president—Mrs. C. P. Gray, Monroe.

Treasurer—Mrs. M. C. Wiginton, Hammond.

Recording Secretary—Mrs. P. A. Donaldson, Reserve.

Corresponding Secretary—Mrs. Herbert Cannon, Covington.

READ THE BULLETIN

The Woman's auxiliary to the American Medical Association is making a special effort at this time to awaken wide-spread interest in its activities, by increasing the number of readers of the *Bulletin*.

This little booklet is a successor to the *News Letter*, which for many years has kept the officers and board members acquainted with the progress of the auxiliary of all the states. It is published quarterly and contains reports of conventions, places of work, inspirational messages from leaders and news of the hour in the medical world. It is a great help in promoting interest in local auxiliaries, especially where the program is new.

The fall issue contains the inaugural address of Mrs. V. E. Holcombe, the National Auxiliary president; also a message to women from Dr. Van Etten, president of the American Medical Association. Many other interesting items are to be found within its forty pages. It is hoped to have 6,000 women, one-fourth of the membership, reading the *Bulletin* before the year is over. In this way the members may keep abreast of the trends in the medical world and be better able to function as members and leaders of the auxiliaries, local, state and national.

The cooperation of the Advisory Councils and the good will of all members of medical associations is earnestly requested.

Mrs. Jules Myron Davidson,
Circulation Chairman.

NEWS OF LOCAL GROUPS

CADDO PARISH

The Woman's Auxiliary to the Shreveport Medical Society held its first meeting of the season Wednesday afternoon, October 9, at the Woman's Department Club.

After a short business session, presided over by the president, Mrs. C. B. Erickson, Mrs. Harold Quinn talked on "Current Events." This was followed by a lovely musical program rendered by Mrs. L. L. Davidge and Miss Dorothy Erickson.

From an artificially appointed table, centered with a silver ship with sail of yellow marigolds on a blue sea of cellophane, coffee and tea were served.

On September 26 and 27, the Shreveport auxiliary was hostess to the Tri-state Medical Society which was held at the Washington-Youree Hotel. Wives of visiting physicians were entertained at a luncheon.

Mrs. W. M. Hall,
Publicity Chairman.

ST. MARY PARISH

The Woman's Auxiliary to the St. Mary Medical Society held its bi-annual meeting on October 12 at Mrs. F. H. Metz's home.

Mrs. T. E. Dreher presided over the meeting and introduced the guest speaker, Mrs. R. Carl Young, president of the state auxiliary, who gave a most interesting address on the aims and projects of the auxiliary. Mrs. A. C. Kappel read an article entitled "A Hundred Years of Medicine."

The auxiliary adopted the following projects, namely, distribution of *Hygeia* in the schools and the donation of two layettes through the Health Center.

Two new members, Mrs. L. J. Supple and Mrs. D. J. Daspit, were introduced.

The officers and chairman of standing committees were presented as follows: president, Mrs. T. E. Dreher; vice-president, Mrs. C. M. Horton; secretary-treasurer, Mrs. F. H. Metz; press and publicity chairman, Mrs. D. J. Daspit.

Mrs. D. J. Daspit,
Publicity Chairman.

SECOND DISTRICT

The Second District Medical Auxiliary met at the home of the president, Mrs. J. Earle Clayton, of Norco, on Tuesday, October 15.

The business meeting was followed by a delightful buffet luncheon.

Mrs. Steve Campbell, *Hygeia* chairman, requests that doctors of the second district send their subscriptions direct to her.

MRS. P. A. DONALDSON,
Publicity Chairman.

Respectfully submitted,
MRS. JONAS W. ROSENTHAL,
Press and Publicity Chairman.

BOOK REVIEWS

Handbook of Skin Diseases: By Leon H. Warren, B. A., M. D., M. Sc. (Med.). New York, Paul B. Hoeber, 1939. Pp. 321. Price $3.50.

This is a synopsis of 252 skin diseases arranged in alphabetical order, with synonyms and eponyms, and cross-indexed for ready reference. The diseases are described in a pithy, concise manner, which aids in diagnosis; principles of therapy are given in a way that makes this little manual a most useful one.

BARRETT KENNEDY, M. D.

The Electrocardiogram in Congenital Cardiac Disease: By Maurice A. Schnitker, B. Sc., M. D. Cambridge, Mass., Harvard University Press, 1940. Pp. 147. Price $3.00.

This book of 130 pages, divided into six chapters, is based on the study of all available instances of congenital heart disease recorded in the literature since 1915 in which an electrocardiogram was illustrated and autopsy findings were available, including 37 cases from the Boston Hospitals. The total series numbered 109 cases: 106 with autopsy reports and one proved by surgical operation.

The cases have been divided into three groups: those without cyanosis, those with late cyanosis, and those with persistent cyanosis. There is a short history of each case, physical and x-ray findings and reproductions of the electrocardiograms interpreted by the author. There are many valuable points on physical signs and differential diagnosis.

With only a very few exceptions can one tell a congenital heart disease from an acquired condition by looking at the electrocardiogram. Dextrocardia with reversed chambers is about the only one.

In this book the author points out that several types of curves would point to congenital heart disease. Marked right axis deviation suggests pulmonary stenosis. The axis deviation seems to be less marked if there is associated interventricular defect, as this lessens the strain on the right ventricle. This bears out the general law of the electrocardiogram that the appearance of the curves will depend upon the chamber of the heart under the greatest strain. There are many possible combinations in congenital heart disease. It was found that the presence of tall pointed P waves or auricular fibrillation in a case of congenital heart disease would point to interauricular septal defect. Seven of the eight cases of congenital heart block studied by the author had interventricular septal defect. In interventricular defects, in addition to conduction defects, curves showing large diphasic QRS complexes are common. QRS complexes of low amplitude with coronary types of T waves in a child or a young adult would make one suspect some anomaly of coronary arteries.

It is interesting to note that 26 per cent of cases reported here showed evidences of rheumatic valvular disease. Hypertension is not common in congenital heart disease except in coarctation of the aorta. Ten cases of this series had pain which was thought to be angina pectoris. The reviewer has seen one case of a five year old child with congenital heart disease in which cardiac pain was strongly suggested.

The reviewer has no hesitation in recommending this book to any one interested in this subject.

J. M. BAMBER, M. D.

A Method of Anatomy; Descriptive and Deductive: By J. C. Boileau Grant, M. C., M. B., Ch. B. Baltimore, The Williams and Wilkins Company, 1940. Pp. xxii + 794; figs. 651. Price $6.00.

Commenting in this Journal on the first edition of Grant's book (1937), the present reviewer characterized it as follows: "An innovation in the field of anatomical text-books. Less than a third of the bulk of the familiar texts of gross anatomy, with original simplified illustrations and above all differing in the method of presenting subject matter, 'A Method of Anatomy' challenges the traditionally plethoric teaching of anatomical rote . . . Emphasis is given to developmental, physiological and practical considerations, and the reader is constantly reminded of significant deductions to be drawn from observed structure." This second edition, like the first, was planned by the author to provide "a working instrument designed to make anatomy rational, interesting and of direct application to the problems of medicine and surgery." It can fulfill that aim even better, with improvements in text matter and illustrations suggested by an experience of three years.

HAROLD CUMMINS, PH. D.

A Handbook for Dissectors: By J. C. Boileau Grant and H. A. Cates. Baltimore, The Williams and Wilkins Company, 1940. Pp. xi + 239; figs. 9. Price $2.50.

This little volume is a guide to procedure in dissection. It is designed for users of the senior author's "A Method of Anatomy," to which the dissector is referred repeatedly by page references to text descriptions and illustrations. Directions for dissection are orderly and clearly stated. Effort is made to distinguish structures of greater and lesser importance by the use of different type faces, bold-face and italics respectively. The nine figures illustrate prescribed incision lines of different regions.

HAROLD CUMMINS, PH. D.

Combined Textbook of Obstetrics and Gynecology: Rev. and rewritten by J. M. Munro Kerr, LL. D., M. D., F. R. F. P. and S. (Glas.), F. R. C. O. G., and others. Baltimore, Williams and Wilkins Co., 1939. Pp. 1192. Price $12.00.

This joint text of obstetrics and gynecology by nine English authors, now in its third edition, has been brought up to date in the latest developments in obstetrics and gynecology, particularly in the endocrine field. All of the recent investigation in this field has been briefly portrayed.

While the combination of these two subjects is desirable, in this instance the gynecologic half of the book is too briefly handled in comparison to the amount of space given to obstetrics. Whereas obstetric procedures are described somewhat in detail, gynecologic surgery is very briefly mentioned.

There is some duplication of information in the book, which the combination obviously had hoped to avoid, e. g., anatomy is considered under two heads and in certain sections endocrinology is considered under different headings. The endocrinologic section is not given as important a role as is found in American texts.

In such a combination of gynecology and obstetrics both fields must necessarily be presented in a more brief manner than in textbooks devoted to the single subjects. For this reason this text is of value to the student but cannot be recommended as a valuable volume for advanced study in either field.

JOHN C. WEED, M. D.

The March of Medicine: Edited by a Committee on Lay Lectures to the Society of the New York Academy of Medicine. New York, Columbia Univ. Press, 1940. Pp. 168. Price $2.00.

The New York Academy of Medicine has for years sponsored a successful series of lay lectures on various phases of the history of medicine. This volume reprints those given in the session 1938-39. It contains, in all, six lectures, three of which this reviewer found to be particularly interesting, namely: "Health in Elizabethan England"; "The Story of Insanity"; and "Not So Long Ago."

This volume is interesting and should appeal to physicians as enjoyable "beside reading." It should also hold the attention of any who have broad interests in the history of medicine.

B. BERNARD WEINSTEIN, M. D.

The Surgery of the Alimentary Tract: By Sir Hugh Devine. Baltimore, Williams and Wilkins Co., 1940. Pp. 1046. Price $15.00.

Any publication by Sir Hugh Devine, who has contributed so much to present day surgery through his advocacy and popularization of dysfunctioning the large bowel as a preliminary to operation on the large intestine, quite naturally attracts attention and deserves consideration. This 1022 page volume is a highly personalized book in which the author presents records and conclusions from his vast clinical experience. It is by no means a complete presentation of the most modern developments and procedures but is rather the author's surgical autobiography. In some sections the material is awkwardly arranged, and although purporting to be a book designed for postgraduates, there is included such elementary matter as knot-tying illustrations. There is a notable paucity of references to the American surgical literature in which there are adequate discussions of many of the developments in surgery which are lacking in this book.

AMBROSE STORCK, M. D.

Minor Surgery: By Frederick Christopher, S. B., M. D., F. A. C. S. Philadelphia, W. B. Saunders Co., 1940. Pp. 990. Price $10.00.

This volume of approximately 1,000 pages, with 639 illustrations, covers adequately what might be classed in the category of minor surgery. It is for that subject almost an exhaustive summary and of necessity teaches many things which certainly, without exaggerating the seriousness of the condition or even the technical difficulties, may be classed under major surgery. The author adequately covers the treatment of wounds and quotes extensively, and fortunately verbatim in many instances, from authors whom he considers representative authorities on the treatment of wounds or other subjects.

Traumatic surgery is discussed fully in the treatment of wounds and fractures. The section on fractures is very good and the illustrations show most modern methods even in the care of fractures of the spine and most of the fractures of the extremity. Open reductions for fractures of the hip are not included.

The reviewer notices with pleasure the very successful effort to keep the book modern. For example, the author has included the latest data on the subject of vascular diseases of the lower extremity. A chapter is devoted to instructions for

the surgical intern with suggestions as to how to manage certain conditions and what he should do when confronted with the necessity of making decisions in the hospital.

One might criticize lightly the chapter on transfusions. It is not on a parity with other chapters. The illustrations showing obsolete methods had better be devoted to illustrations of the direct and indirect methods which are in common use in most hospitals today. It is true that such methods as the vacoliter flask are illustrated with acknowledgment given to the proper medical author; nevertheless, similar apparatus has been perfected by certain commercial houses and is in general use, so much so, that an intern in most cities would probably encounter a Baxter transfusion set more readily than a similar flask made by the hospital itself. The direct methods of transfusion illustrated, namely the Scannel and the direct paraffin lined tube with a pressure bulb, when compared to some of the more modern transfusion sets for direct transfusion are of historical interest and even then of only minor historical interest as far as transfusions are concerned.

The student and practitioner will gain reliable worthwhile information from this book. It will be of value in the management of the majority of cases of a surgical nature which the general practitioner might wish to handle.

HOWARD MAHORNER, M. D.

Graduate Medical Education: Report of the Commission on Graduate Medical Education. Chicago, University of Chicago Press, 1940. Pp. 304. Gratis.

This volume is a report of the Commission on Graduate Medical Education which was organized on December 4, 1937. It undertakes to "formulate the educational problems and principles involved in the continuation of medical training for a period of years after graduation and the adequate training of specialists, and to make recommendation for methods whereby those in practice, general and limited, may keep abreast of new developments in diagnosis, treatment and prevention."

The internship, the residency, specialism, the specialty boards, postgraduate education (intra- and extramural) and other pertinent subjects are thoroughly covered. The book is replete with valuable suggestions and food for thought, and is delightfully "readable." While some important efforts in these fields are not even mentioned, on the whole the subject is very well covered.

Everyone interested in "continuation study" for physicians, in any of its many forms, should study this report. Logically, this would include every physician in this country, but should by all means include all teachers of medicine, whether undergraduate, graduate, or postgraduate.

H. W. KOSTMAYER, M. D.

Psychiatric Clinics for Children, with Special Reference to State Programs: By Helen Leland Witmer, Ph. D. New York, Commonwealth Fund, 1940. Pp. 437. Price $2.50.

The author of this excellent, complete, authoritative sourcebook is a distinguished scholar and an educator. She has utilized a vast amount of valuable material and has expended much well directed effort in analyzing it and estimating its intrinsic worth and practical applicability.

To provide necessary information for those desiring to make child guidance clinic service available throughout the country, The National Committee for Mental Hygiene arranged for a study and an appraisal of programs offering such service on a state-wide basis. A review of the philosophy, objectives, practices and achievements of clinical child psychiatry was required. The results are presented by Dr. Witmer in this volume.

The subject matter of "Psychiatric Clinics for Children" is offered in three parts. Part I describes the theoretical and historical background of clinical child psychiatry in the United States and the cultural setting in which it operates. Part II is concerned with a survey of state-financed clinics. It tells how they are conducted and describes and evaluates typical policies. Part III discusses principles for future programs. It suggests that many of the difficulties of such clinics are traceable to lack of well-defined objectives and examines the alternative possibilities in the light of modern psychiatric theory and practice.

This treatise is a notable contribution in the field of clinical psychiatry. It has great merit and presents all angles of the stiuation clearly and concisely. It deserves the attention, interest and approval of those who plan and organize clinics for children and who would understand the general theory and problems of child guidance.

The book ends with a list of basic requirements which, experience has proved, must be met if a program of clinical psychiatry is to be successful and with the statement that "mental hygiene to be most effective must be reflected in a way of life in the community as a whole."

C. P. MAY, M. D.

Differential Diagnosis in Internal Medicine: By Otto Naegeli. Chicago, S. B. Debour, 1940. Pp. 726. Price $10.00.

This posthumous work by a famous physician should be of interest to all internists. The language and phraseology of the translation tends to make the book a little difficult reading in places. One is also struck by the lack of references to American and English authors. The most fascinating thing about the book is the personal element. It is an expression of the author's own wide knowledge and experience. Throughout the work the author inserts short case histories illustrating certain points in differential diagnosis. His method of arriving

at a diagnosis is not by a process of simple elimination but rather by dividing diseases into groups; for example inflammations, intoxications, malignant tumors, systemic hyperplasias of the lymphatic or myeloid apparatus. After the offending group has been determined upon, then the differentiation is made between the different members or diseases found in the group. How extensive Professor Naegeli's experience has been is demonstrated by the fact that the book takes up some 700 odd pages. The reviewer recommends it to all physicians interested in differential diagnosis.

RANDOLPH LYONS, M. D.

Clinical Heart Disease: By Samuel A. Levine, M. D., F. A. C. P. Philadelphia, W. B. Saunders Company, 1940. Pp. 495. Price $6.00.

The second edition of this popular book contains all of the material present in the first (1936) and has been enlarged by the addition of one chapter, 64 pages of text, 12 figures, and by amplification of the index.

The new material covers briefly but adequately the recent advances in cardiology. It includes, as well, a chapter on the medico-legal aspects of heart disease, a sub-chapter on the nature of circulatory failure, another sub-chapter on the rarer forms of heart disease, such as beri-beri heart, cor pulmonale, and others, several pages on the action of digitalis, and a description of the new approved technic and nomenclature for the precordial lead.

Those familiar with the recommendations contained in the first edition will be interested to learn that the author now rarely advises thyroidectomy for angina pectoris and that he only occasionally prescribes quinidine following acute myocardial infarction. Injections of mercury are still advocated in the treatment of syphilitic heart disease, and no mention is made of bismuth.

There are several improvements of a general nature noted in the new edition. The text actually begins on page 1, rather than on page 17, as in the first edition. The first person pronoun, "I" appears less frequently. Phraseology, and sentence and paragraph construction are better, although grammarians may still find fault with the use of nouns in place of adjectives (as in "mercury diuretics") and with the use of "finally" to introduce several different paragraphs in the same chapter.

It is gratifying to find that wherever the first edition reserved judgment until further experience had accumulated, the second edition nearly always contains a progress note bringing the matter up to date. However, in spite of a four year interval between editions, the words, "recent" and "recently," in many statements have been carried over into the second edition unchanged. This fault is always annoying to the reader and may be carried to such ridiculous extremes as in one standard text, the 1931 edition of which contains the expression "recently" with a footnote dating the reference 1903.

Although a new edition of this work is welcome, the old edition is by no means rendered obsolete, for the very best chapters remain essentially unaltered. In no other book on heart disease can one find so clearly portrayed such subjects as the significance of the systolic murmur, prognosis in heart disease, the patient with heart disease as a surgical or obstetrical risk, paroxysmal rapid heart action, and the correlation of clinical and electrocardiographic findings.

This book is highly recommended to all who wish to improve their knowledge of clinical heart disease.

EDWARD MATTHEWS, M. D.

Congenital Cleft Lip, Cleft Palate and Associated Nasal Deformities: By Harold Stearns Vaughan, M. D., D. D. S., F. A. C. S. Philadelphia, Lea and Febiger, 1940. Pp. 210. Price $4.00.

This compact volume is a valuable contribution because it includes the basic fundamentals involved in the proper treatment of two distressing congenital defects: cleft lip and cleft palate. This edition includes and brings up to date the worth-while developments in treatment and the refinements of operative technic which are of proved value. The author does not go into too much detail in specific procedures for the book apparently is designed to be of practical reference value to the general surgeon and the pediatricians.

NEAL OWENS, M. D.

Obesity and Leanness: By Hugo R. Rony, M. D. Philadelphia, Lea & Febiger, 1940. Pp. 300. Price $3.75.

This is an excellent and comprehensive review of this subject. The author expounds his feeling that the subject is not one of calories and metabolism alone, but a much more complicated affair.

The book is divided into three sections, namely, a synopsis of the physiology of fats and fat tissue, the pathogenesis of obesity and leanness, and the clinical aspects of obesity and leanness.

To anyone interested in medicine, this is a clear presentation of the pathology and clinical aspects of the subject. It is a pleasure to read a book on obesity and leanness without having to wade through long lists of diets; the reader feels that every angle has been elucidated.

SUZANNE SCHAEFER, M. D.

PUBLICATIONS RECEIVED

F. A. Davis Company, Philadelphia: Taber's Cyclopedic Medical Dictionary, by Clarence Wilbur Taber and fourteen associates.

The Williams & Wilkins Company, Baltimore: Fractures and Dislocations, by Edwin O. Geckeler, M. D.